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Static and Dynamic Pressure-Volume Curves in Experimental ARDS

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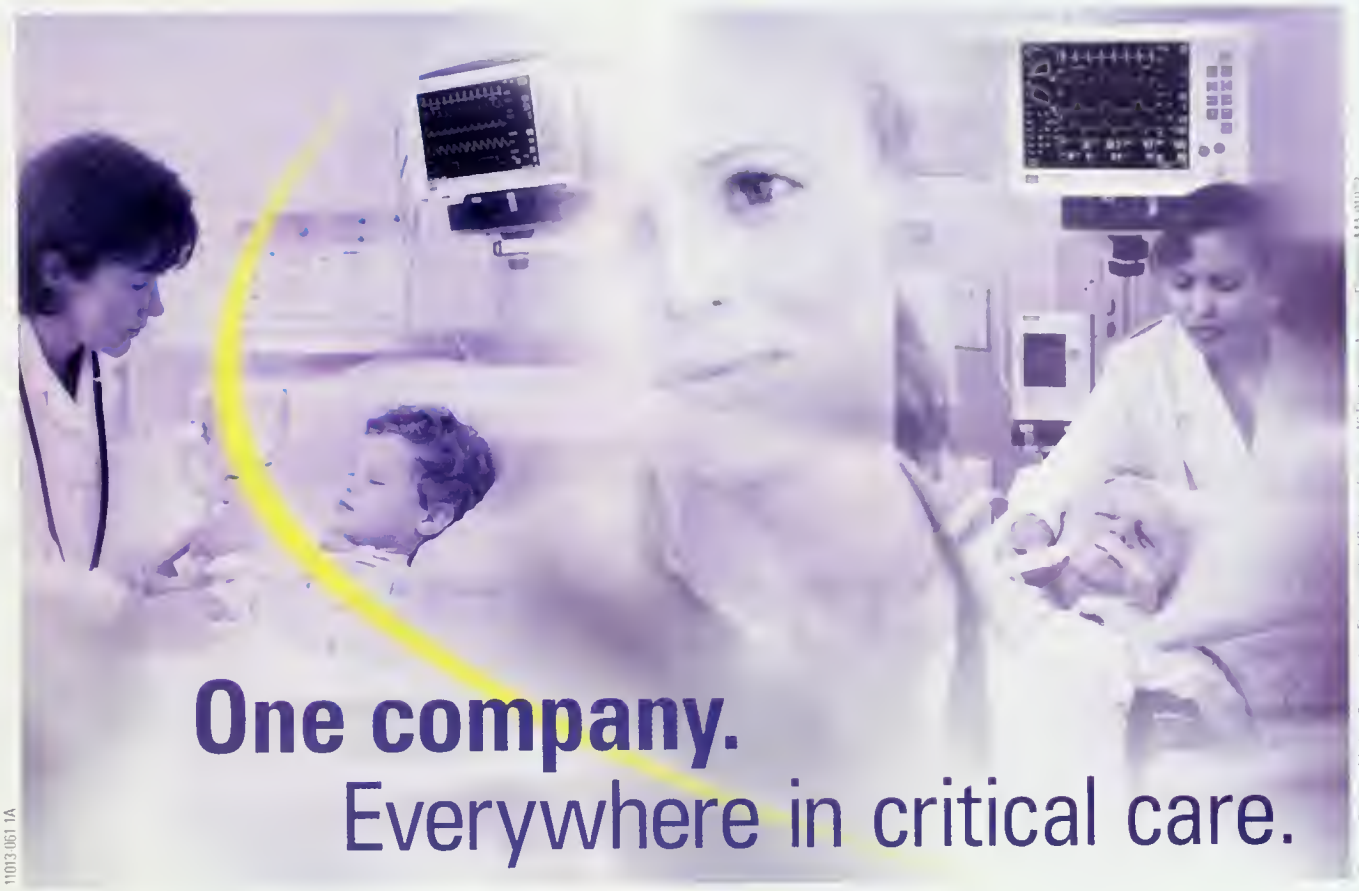

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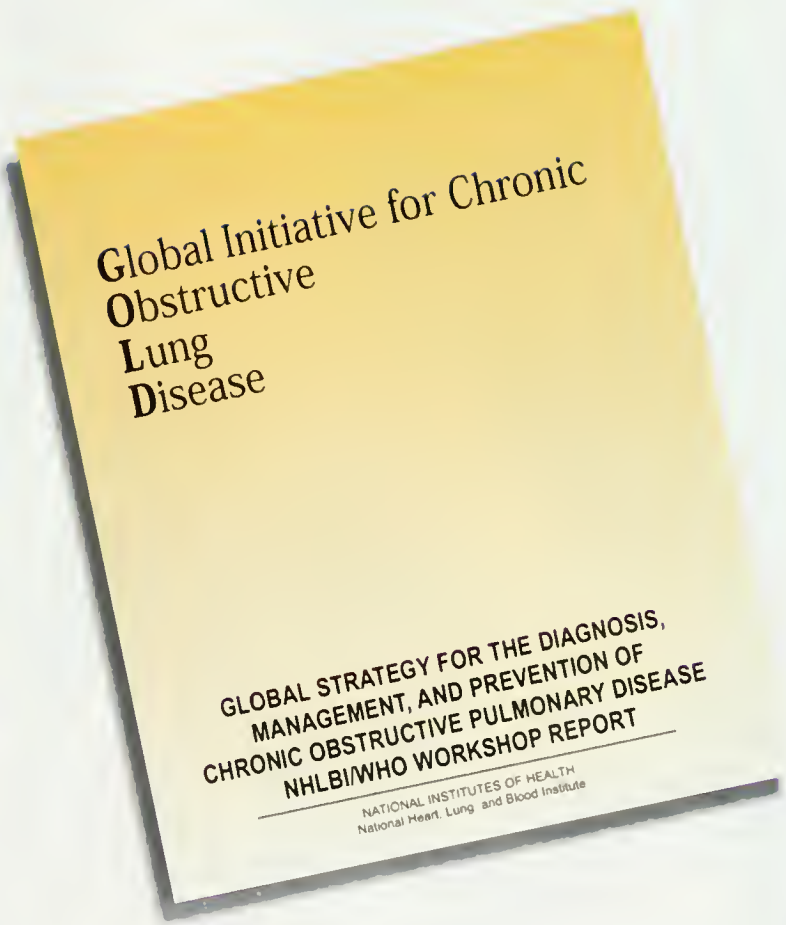
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Editorials, Commentaries, and Reviews To Note

The Evidence Base for Management of Acute Exacerbations of COPD: Clinical Practice Guideline, Part 1—Snow V, Lascher S, Mottur-Pilson C. Chest 2001;119(4):1185-1189.

Antibiotics for Acute Bronchitis (editorial)—Arroll B, Kenealy T. BMJ 2001;322(7292):939-940.

Considerations in the Relationship Between the American College of Chest Physicians and Industry (comment)—Lever A. Chest 2001;119(4):1257-1259.

Physician Education and the Pharmaceutical Industry (editorial)—Johnson RG. Chest 2001; 119(4):995-996.

Pulmonary Artery Catheterization in the ICU/Critical Care Unit : Indications and Contraindications Remain Objectively Undefined (editorial)—Spodick DH. Chest 2001;119(4): 999-1000.

Life-Threatening Hypoxemic Respiratory Failure After Repair of Acute Type A Aortic Dissection: Successful Treatment with Venoarterial Extracorporeal Life Support Using a Prosthetic Graft Attached to the Right Axillary Artery—Yokota K, Fujii T, Kimura K, Toriumi T, Sari A. Anesth Analg 2001;92(4):872-876.

In Search of a Reliable Awareness Monitor (editorial)—Tempe DK. Anesth Analg 2001;92(4): 801-804.

Pulmonary Blood Flow Distribution in Stage 1 Chronic Obstructive Pulmonary Disease—Capderou A, Aurengo A, Derenne JP, Similowski T, Zelter M. Am J Respir Crit Care Med 2000 Dec;162(6):2073–2078.

We investigated the hypothesis that lung blood flow distribution is modified in stage 1 chronic obstructive pulmonary disease (COPD). We compared patients with stage 1 COPD (n = 11) with restrictive patients with comparable blood gases (n = 7), to patients with low cardiac index with normal lungs (n = 11) and to control subjects (n = 11). Distribution of transit time (DTT) was computed by deconvolution from first pass radioactivity curves (albumin ^{99m}Tc) reconstructed from right and left ventricular regions of interest. Distribution descriptors, mean transit time ($p < 0.05$), standard deviation ($p < 0.001$), relative dispersion ($p < 0.001$), and kurtosis ($p < 0.001$) differed between groups (ANOVA). Cardiac index was the same in COPD and low CI groups but lower compared with normal subjects ($p < 0.05$). After normalization for cardiac output, the DTT of patients with COPD remained different from low CI and restrictive patients ($p < 0.001$). Therefore changes in DTT in patients with COPD compared with patients without COPD could not be explained on the basis of difference in cardiac output. Because P_{O2}, P_{CO2}, and pH were similar in COPD and restrictive groups, difference in distribution could not be explained either on the basis of blood gas data. We conclude that changes in DTT occurs in stage 1 COPD and cannot be explained by hypoxemia, hypercapnia, or acidosis alone but must relate to other structural or regulatory responses.

Gender-Related Differences in Severe, Early-Onset Chronic Obstructive Pulmonary Disease—Silverman EK, Weiss ST, Drazen JM, Chapman HA, Carey V, Campbell EJ, et al. Am J Respir Crit Care Med 2000 Dec;162(6):2152–2158.

Men have higher prevalence rates of chronic obstructive pulmonary disease (COPD) than women, which has been attributed to the historically higher rates of cigarette smoking in males. However, the increased rates of cigarette smoking in females within the last several decades have been associated with steadily increasing rates of COPD in women. As part of a study of the genetics of severe, early-onset COPD, we assembled a group of 84 probands with severe, early-onset COPD (without severe alpha$_1$-antitrypsin deficiency) and 348 of their first-degree relatives. We found a markedly elevated prevalence (71.4%) of females among the early-onset COPD probands. Among the entire group of first-degree relatives of early-onset COPD probands, univariate analysis demonstrated similar spirometric values and bronchodilator responsiveness in males and females; however, among current or ex-smokers, female first-degree relatives had significantly lower FEV_1/ FVC (81.4 ± 17.2% in females versus 87.0 ± 12.9% in males, $p = 0.009$) and significantly greater bronchodilator responsiveness (expressed as percentage of baseline FEV_1) (7.7 ± 9.4% pred in females versus 4.1 ± 6.4% pred in males, $p = 0.002$). Female smoking first-degree relatives were significantly more likely to demonstrate profound reductions in FEV_1 (< 40% pred) than male smoking first-degree relatives ($p = 0.03$). Multivariate analysis, performed with generalized estimating equations, demonstrated that current or ex-smoking female first-degree relatives had significantly greater risk of $FEV_1 < 80\%$ pred (OR 1.91, 95% CI 1.03- 3.54), $FEV_1 < 40\%$ pred (OR 3.56, 95% CI 1.08–11.71), and bronchodilator response greater than 10% of baseline FEV_1 (OR 4.74, 95% CI 1.91–11.75). These results

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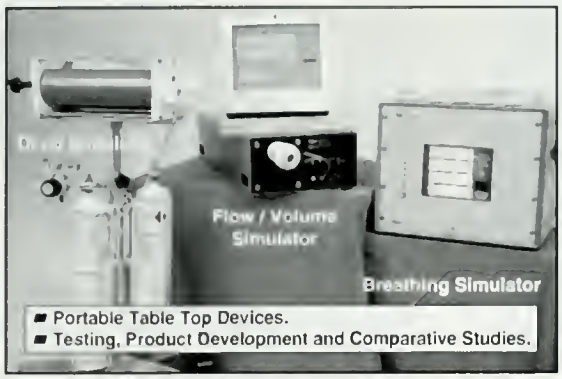

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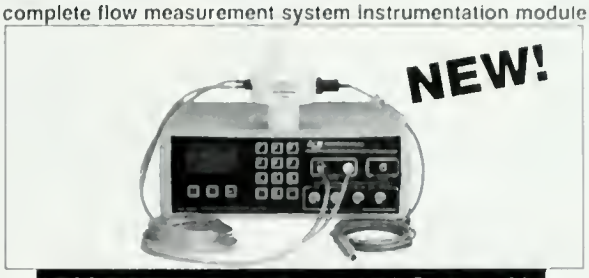

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suggest that women may be more susceptible to the development of severe COPD.

Effectiveness of Oseltamivir in Preventing Influenza in Household Contacts: A Randomized Controlled Trial—Welliver R, Monto AS, Carewicz O, Schatteman E, Hassman M, Hedrick J, et al. JAMA 2001 Feb 14;285(6):748–754.

CONTEXT: Influenza virus is easily spread among the household contacts of an infected person, and prevention of influenza in household contacts can control spread of influenza in the community. OBJECTIVE: To investigate the efficacy of oseltamivir in preventing spread of influenza to household contacts of influenza-infected index cases (ICs). DESIGN AND SETTING: Randomized, double-blind, placebo-controlled study conducted at 76 centers in North America and Europe during the winter of 1998–1999. PARTICIPANTS: Three hundred seventy-seven ICs, 163 (43%) of whom had laboratory-confirmed influenza infection, and 955 household contacts (aged ≥12 years) of all ICs (415 contacts of influenza-positive ICs). INTERVENTIONS: Household contacts were randomly assigned by household cluster to take 75 mg of oseltamivir (n = 493) or placebo (n = 462) once daily for 7 days within 48 hours of symptom onset in the IC. The ICs did not receive antiviral treatment. MAIN OUTCOME MEASURE: Clinical influenza in contacts of influenza-positive ICs, confirmed in a laboratory by detection of virus shedding in nose and throat swabs or a 4-fold or greater increase in influenza-specific serum antibody titer between baseline and convalescent serum samples. RESULTS: In contacts of an influenza-positive IC, the overall protective efficacy of oseltamivir against clinical influenza was 89% for individuals (95% confidence interval [CI], 67%–97%; p<0.001) and 84% for households (95% CI, 49%–95%; p<0.001). In contacts of all ICs, oseltamivir also significantly reduced incidence of clinical influenza, with 89% protective efficacy (95% CI, 71%–96%; p<0.001). Viral shedding was inhibited in contacts taking oseltamivir, with 84% protective efficacy (95% CI, 57%–95%; p<0.001). All virus isolates from oseltamivir recipients retained sensitivity to the active metabolite. Oseltamivir was well tolerated; gastrointestinal tract effects were reported with similar frequency in oseltamivir (9.3%) and placebo (7.2%) recipients. CONCLUSION: In our sample, postexposure prophylaxis with oseltamivir, 75 mg once daily for 7 days, protected close contacts of influenza-infected persons against influenza illness, prevented outbreaks within households, and was well tolerated.

Diagnostic Accuracy of a Bedside D-Dimer Assay and Alveolar Dead-Space Measurement for Rapid Exclusion of Pulmonary Embolism: A Multicenter Study—Kline JA, Israel EG, Michelson EA, O'Neil BJ, Plewa MC, Portelli DC. JAMA 2001 Feb 14;285(6):761–768.

CONTEXT: A previous study suggested that the combination of a normal D-dimer assay and normal alveolar dead-space fraction is a highly sensitive screening test for pulmonary embolism (PE). OBJECTIVE: To determine if the combination of a normal alveolar dead-space fraction (volume of alveolar dead space/tidal volume ≤20%) and a normal whole-blood agglutination D-dimer assay can exclude PE in emergency department (ED) patients. DESIGN: Prospective, noninterventional study conducted in 1998–1999. Study data were obtained prior to standard testing for PE, consisting of radionuclide lung scanning or contrast-enhanced computed tomography and 6-month follow-up plus selective use of venous ultrasonography and pulmonary angiography. Imaging studies were interpreted by blinded observers. SETTING: Six urban teaching hospitals in the United States. PATIENTS: A total of 380 hemodynamically stable ED patients aged 18 years or older with suspected acute PE. MAIN OUTCOME MEASURES: Sensitivity and specificity for PE with a positive test defined as having either alveolar dead-space fraction or D-dimer assay results abnormal. Alveolar dead-space fraction was determined by

subtracting airway dead space from physiological dead space (determined using the modified Bohr equation) and D-dimer assay, assayed at bedside using 20 microL of arterial blood. RESULTS: Pulmonary embolism was diagnosed in 64 patients (16.8%), of those 20 had an abnormal D-dimer assay result, 3 had an abnormal alveolar dead-space fraction, 40 had abnormal results in both, and 1 had normal results for both tests. The sensitivity for diagnosis of PE was 98.4% (95% confidence interval [CI], 91.6%–100.0%). Among the 316 patients without PE, both D-dimer and dead-space results were normal in 163, for a specificity of 51.6% (95% CI, 46.1%–57.1%). Posterior probability of PE with normal results on both tests was 0.75% (95% CI, 0%–3.4%). CONCLUSION: In this multicenter study of ED patients, a normal D-dimer assay result plus a normal alveolar dead-space fraction was associated with a low prevalence of PE.

Quality of Life in Patients Awaiting Lung Transplant: Cystic Fibrosis Versus Other End-Stage Lung Diseases—Burker EJ, Carels RA, Thompson LF, Rodgers L, Egan T. Pediatr Pulmonol 2000 Dec;30(6): 453–460.

The symptoms associated with chronic lung disease can impair quality of life and psychosocial functioning. The purpose of the present study was to provide a thorough baseline assessment of quality of life in patients with end-stage lung disease and being evaluated for transplant; and to assess potential differences in quality of life between patients with cystic fibrosis (CF) and those with other types of end-stage lung disease (e.g., chronic obstructive pulmonary disease (COPD), interstitial pulmonary fibrosis (IPF)). We evaluated 58 patients with CF and 52 patients with other types of end-stage lung disease who were recruited for this study during an assessment of their candidacy for lung transplant. Subjects completed a battery of questionnaires that assessed demographic factors (including work and educational status), the presence of psychological distress (anxiety and depression), availability of social support, coping styles, and physical functioning. Despite significant impairment in physical functioning in the areas of recreation, household activities, sleep, and ambulation, other indices of life quality suggested good adaptation in the majority of patients. Also, quality of life differed for patients with CF and for those with other types of end-stage lung disease. Patients with CF were more likely to be working, had lower levels of anxiety and higher levels of social support, and used more functional coping strategies than did patients with other end-stage lung disease. These results highlight the fact that patients with different types of lung disease may require different psychosocial services as they await transplant. These findings also raise the question of whether there is a difference in quality of life after transplant between patients with CF and those with other types of lung disease.

Self-Hypnosis for Patients with Cystic Fibrosis—Anbar RD. Pediatr Pulmonol 2000 Dec;30(6):461–465.

This report documents the utility of self-hypnosis in patients with cystic fibrosis (CF). Sixty-three patients 7 years of age or older were offered the opportunity to be taught self-hypnosis by their pulmonologist. Forty-nine agreed to learn it. Patients generally were taught hypnosis in one or two sessions. The outcome of hypnotherapy was determined by patients' answers to open-ended questions regarding their subjective evaluation of the efficacy of hypnosis. The average age of the 49 patients who were taught and used self-hypnosis was 18.1 years (range, 7–49 years). Many of the patients used hypnosis for more than one purpose, including relaxation (61% of patients), relief of pain associated with medical procedures (31%), headache relief (16%), changing the taste of medications to make the flavor more palatable (10%), and control of other symptoms associated with CF (18%). The patients successfully utilized self-hypnosis 86% of the time. No symptoms worsened following hypnotherapy. Sixteen patients chose to practice hypnosis on their own for a half year or longer. In conclusion, with the use of self-hypnosis, patients with CF can quickly learn to enhance their control over discomforts associated with therapy and their disease. Consideration should be given to making instruction in self-hypnosis available to patients with CF.

Accumulation of CO_2 in Reservoir Devices During Simulated Neonatal Mechanical Ventilation—Lugo RA, Keenan J, Salyer JW. Pediatr Pulmonol 2000 Dec;30(6):470–475.

Aerosolized albuterol is frequently administered to mechanically ventilated neonates by metered dose inhaler (MDI) and a reservoir device. These reservoirs are often placed between the Y-piece and endotracheal tube, thereby creating mechanical dead space and increasing the risk of rebreathing carbon dioxide (CO_2). The objectives of this study were: 1) to quantify CO_2 accumulation in two commonly used reservoirs (ACE®, Aerochamber®-MV) and a bidirectional nonreservoir actuator (Airlife® Minispacer) during mechanical ventilation of a neonatal lung model; and 2) to determine the effect of tidal volume (V_T) on CO_2 accumulation. We hypothesized that the accumulation of CO_2 in these devices is clinically insignificant at the small tidal volumes used in mechanically ventilated premature neonates. The model was constructed to simulate CO_2 exhalation by a ventilated neonate and consisted of a neonatal ventilator circuit (rate = 40/min; peak inspiratory pressure (PIP) = 20 cm H_2O) attached to a reservoir/actuator and neonatal test lung. The ventilator delivered inspiratory gas (room air) to the test lung, which was vented into the atmosphere by a small adjustable leak. Expiration was simulated by manually ventilating 7.1% CO_2 (partial pressure of CO_2 (P_{CO_2}) = 48 mm Hg) back through the model. Accumulation of CO_2 within the reservoir/actuator was measured using an end-tidal CO_2 monitor. Each 4-min experiment was conducted at three V_T (7.5 mL, 15 mL, and 25 mL), and the median P_{CO_2} was calculated in 0.5-min increments. For V_T = 7.5 mL, CO_2 accumulated slowly in the ACE and Minispacer and reached a maximum at 4.0 min (P_{CO_2} = 2.3 mm Hg and 7.3 mm Hg, respectively). In contrast, the Aerochamber-MV rapidly reached a P_{CO_2} of 9.5–10.0 mm Hg by 1–1.5 min. A similar trend occurred with V_T = 15 mL; however, higher partial pressures (approximately 10–12 mm Hg) were achieved with all devices. At V_T = 25 mL, P_{CO_2} rose rapidly with the ACE, Aerochamber-MV, and Minispacer, reaching peaks of 17.2, 12.3, and 20.3 mm Hg, respectively ($p < 0.05$). In conclusion, accumulation of CO_2 in reservoir/actuator depends on V_T as well as the chamber design and internal volume. Due to the short duration of use when administering drugs via MDI, accumulation of CO_2 in these devices is not likely to be clinically relevant for the majority of ventilated newborns.

Noninvasive Ventilation in Immunosuppressed Patients with Pulmonary Infiltrates, Fever, and Acute Respiratory Failure—Hilbert G, Gruson D, Vargas F, Valentino R, Gbikpi-Benissan G, Dupon M, et al. N Engl J Med 2001 Feb 15;344(7):481–487.

BACKGROUND: Avoiding intubation is a major goal in the management of respiratory failure, particularly in immunosuppressed patients. Nevertheless, there are only limited data on the efficacy of noninvasive ventilation in these high-risk patients. METHODS: We conducted a prospective, randomized trial of intermittent noninvasive ventilation, as compared with standard treatment with supplemental oxygen and no ventilatory support, in 52 immunosuppressed patients with pulmonary infiltrates, fever, and an early stage of hypoxemic acute respiratory failure. Periods of noninvasive ventilation delivered through a face mask were alternated every three hours with periods of spontaneous breathing with supplemental oxygen. The ventilation periods lasted at least 45 minutes. Decisions to intubate were made according to standard, predetermined criteria. RESULTS: The base-line characteristics of the two groups were similar; each group of 26 patients included 15 patients with hematologic cancer and neutropenia. Fewer patients in the noninvasive-ventilation group than in the standard-treatment group required endotra-

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Invasive Ventilation: The Latest Word

Richard Kallet, MS, RRT, and Richard D. Branson, BA, RRT, FAARC

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cheal intubation (12 vs. 20, p=0.03), had serious complications (13 vs. 21, p=0.02), died in the intensive care unit (10 vs. 18, p=0.03), or died in the hospital (13 vs. 21, p=0.02). CONCLUSIONS: In selected immunosuppressed patients with pneumonitis and acute respiratory failure, early initiation of noninvasive ventilation is associated with significant reductions in the rates of endotracheal intubation and serious complications and an improved likelihood of survival to hospital discharge.

Withholding and Withdrawal of Life Support in Intensive-Care Units in France: A Prospective Survey. French LATAREA Group—Ferrand E, Robert R, Ingrand P, Lemaire F. Lancet 2001 Jan 6;357(9249): 9–14.

BACKGROUND: In France, there are no guidelines available on withholding and withdrawal of life-sustaining treatments, and information on the frequency of such decisions is scarce. METHODS: We undertook a prospective 2-month survey in 113, of a total of 220, intensive-care units (ICUs) in France to study the frequency of, and processes leading to, decisions to withhold and withdraw life-sustaining treatments. FINDINGS: Life-supporting therapies were withheld or withdrawn in 807 (11.0%) of 7309 patients (withholding in 336 [4.6%] and withdrawal in 471 [6.4%], preceded in 358 by withholding). Of 1175 deaths in ICU, 628 (53%) were preceded by a decision to limit life-supporting therapies. Futility and poor expected quality of life were the most frequently cited reasons. Decisions were strongly correlated with the simplified acute physiological score, but an independent centre effect persisted after adjustment for this score. Decisions were mostly taken by all the ICU medical staff, with (54%) or without (34%) the nursing staff; however, a single physician made decisions in 12% of cases. The patient's family was involved in the decision-making process in 44% of cases. The patient's willingness to limit his or her own care was known in only 8% of the cases; only 0.5% of the patients were involved in decisions. INTERPRETATION: Withholding and withdrawal of life-support therapies are widely practised in French ICUs, despite their prohibition by the French legislation. The lack of an official statement from French scientific bodies may explain several limitations on the various steps of the decision-making process.

A Randomized, Double-Blinded Comparison of Intrathecal Morphine, Sufentanil and Their Combination Versus I.V. Morphine Patient-Controlled Analgesia for Postthoracotomy Pain—Liu N, Kuhlman G, Dalibon N, Moutafis M, Levron JC, Fischler M. Anesth Analg 2001 Jan;92(1):31–36.

We compared the analgesic effect of lumbar intrathecal (IT) 0.5 mg morphine (Group M, n = 10), 50 microg sufentanil (Group S, n = 10), and their combination (Group S-M, n = 10) given before general anesthesia and patient-controlled analgesia with I.V. morphine (Group C, n = 19) in a randomized, double-blinded study performed in patients undergoing thoracotomy. Pain visual analog scale (VAS) and morphine consumption were assessed for 24 h. In Group S-M the number of patients initially titrated with I.V. morphine was less than in group C (30 vs 84%, $p < 0.05$). Morphine requirement was higher in Group C (71 ± 30 mg) than in Groups S (46 ± 34 mg, $p < 0.05$), M (38 ± 31 mg, $p < 0.05$) and S-M (23 ± 16 mg, $p < 0.01$). VAS scores were significantly decreased during the first 0–11 postoperative h at rest and during the first 0–8 postoperative h on coughing in Groups M and S-M rather than in Group C. The incidence of side effects was infrequent except for urinary retention. Preoperative IT morphine or combined sufentanil and morphine could be given as a booster to achieve rapidly effective analgesia in the immediate postoperative period. Implications: As compared with I.V. patient-controlled analgesia, intrathecal morphine or combined sufentanil and morphine provided superior postoperative pain relief both at rest (11 h) and on coughing (8 h) than did I.V. patient-controlled analgesia morphine alone. I.V. morphine requirement was decreased during the first postoperative day after posterolateral thoracotomy.

Volume Ventilation of Infants with Congenital Heart Disease: A Comparison of Dräger, NAD 6000 and Siemens, Servo 900C Ventilators—Stayer SA, Andropoulos DB, Bent ST, McKenzie ED, Fraser CD. Anesth Analg 2001 Jan;92(1):76–79.

We compared the ventilation and pulmonary mechanics produced by a new anesthesia ventilator (NAD 6000) using a circle system with that produced by a critical care ventilator (Servo 900C) using a nonrebreathing circuit in infants with congenital heart disease. Twenty patients, aged 1 day to 7 mo, weighing 2.1 to 4.6 kg, were studied. The NAD 6000 had improved alveolar ventilation: PaCO(2) 43 ± 8 vs 47 ± 5 mm Hg ($p = 0.005$), end-tidal CO_2 34 ± 7 vs 37 ± 5 mm Hg ($p = 0.042$); larger inspired tidal volumes 12.9 ± 2.8 vs 11.3 ± 2.2 mL/kg ($p < 0.001$), but with higher mean airway pressures 9.7 ± 1.6 vs 8.6 ± 1.3 cm H_2O ($p < 0.001$). These differences in ventilation and airway pressures were not clinically significant. Although there were differences in observed ventilatory variables, both machines provided adequate ventilation when set in the volume control mode. Implications: We compared two ventilators for use in infants. Twenty infants undergoing surgery for congenital heart defects were randomized to receive ventilation first with one ventilator, then with the other. Although there were differences in observed ventilatory variables, both machines provided adequate ventilation when set in the volume control mode.

The Effect of Breath Termination Criterion on Breathing Patterns and the Work of Breathing During Pressure Support Ventilation—Tokioka H, Tanaka T, Ishizu T, Fukushima T, Iwaki T, Nakamura Y, Kosogabe Y. Anesth Analg 2001 Jan;92(1):161–165.

With pressure support ventilation (PSV), each PSV breath is flow-cycled, and the breath termination criterion (TC) is usually nonadjustable. When TC does not match the interaction between the patient's inspiratory-expiratory efforts to the opening and closing of the inspiratory and expiratory valves, patient-ventilator asynchrony may occur, and the work of breathing (WOB) may increase. Therefore, we studied the effect of TC on breathing patterns and WOB during PSV in eight patients with acute respiratory distress syndrome or acute lung injury. We studied five levels of TC during PSV-1%, 5%, 20%, 35%, and 45% of the peak inspiratory flow. With increasing levels of TC, the tidal volume decreased and respiratory frequency increased, along with a decrease in duty cycle. WOB markedly increased with increasing levels of TC from 0.31 ± 0.12 J/L with TC 1% to 0.51 ± 0.11 J/L with TC 45%. Premature termination with double breathing occurred in one patient with TC 35% and four patients with TC 45%. Delayed termination with a duty cycle of >0.5 occurred in two patients with TC 1%. In conclusion, the proper adjustment of TC improves patient-ventilator synchrony and decreases WOB during PSV. Implications: Although termination criterion (TC) is usually nonadjustable, it influences the effectiveness of pressure support ventilation for mechanical ventilation. The proper adjustment of TC is crucial to improve patient-ventilator synchrony and decrease work of breathing. TC 5% of the peak inspiratory flow may be the optimal value for patients with acute respiratory distress syndrome or acute lung injury.

Negative Pressure Rewarming vs. Forced Air Warming in Hypothermic Postanesthetic Volunteers—Taguchi A, Arkilic CF, Ahluwalia A, Sessler DI, Kurz A. Anesth Analg 2001 Jan;92(1):261–266.

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a tympanic membrane temperature near 34 degrees C. Anesthesia was discontinued and shivering was prevented by using meperidine. The vasoconstricted volunteers were rewarmed for 2 h using three randomly assigned methods: 1) Vital Heat plus cotton blanket; 2) one layer of cotton blanket; 3) forced-air warming. Thermal flux was recorded from 15 skin-surface sites; metabolic heat production was estimated from total body oxygen consumption. Metabolic heat production remained constant throughout the study. Systemic heat loss remained constant during warming with cotton blankets but decreased significantly during the other treatments. Systemic heat balance increased significantly more with forced air (140 ± 21 kcal) than with Vital Heat (66 ± 19 kcal) or cotton blankets (47 ± 18 kcal). Core temperature increased no faster with Vital Heat warming (1.3 ± 0.4 degrees C) than with a cotton blanket (1.2 ± 0.4 degrees C). In contrast, core temperature increased more rapidly with forced air warming (2.6 ± 0.6 degrees C). In this study we show that calories from a negative pressure rewarming device are largely constrained to the forearm and that heat does not flow to the core thermal compartment.

A Videographic Analysis of Laryngeal Exposure Comparing the Articulating Laryngoscope and External Laryngeal Manipulation—Ochroch EA, Levitan RM. Anesth Analg 2001 Jan;92(1):267–270.

Activation of the articulating laryngoscope and external laryngeal manipulation (ELM) improve laryngeal exposure during direct laryngoscopy. We used a head-mounted direct laryngoscopy imaging system and a previously validated scoring system for assessing laryngeal view (the percentage of glottic opening or POGO score) on 33 adult patients undergoing laryngoscopy. On each patient, we videotaped the initial laryngeal exposure (blade not activated), the view with activation of the blade, and the view with operator-directed external laryngeal manipulation. The video recordings were reviewed and the laryngeal view assessed with POGO scores. POGO scores improved with blade activation in 9/33 (27%) of patients vs 28/33 (85%) of patients with ELM. In nearly half of patients studied (16/33, 48%) POGO scores decreased with blade activation. We conclude that ELM is superior to articulating laryngoscope blade activation in improving POGO scores during laryngoscopy on adult patients in standard sniffing position. Using recordings from a direct laryngoscopy video system, we compared laryngeal views in 33 patients with a special articulating laryngoscope blade to views achieved by external laryngeal manipulation (pressing on the patient's neck). Laryngeal exposure, which is important for placement of tracheal tubes, was better with external laryngeal manipulation.

Early Diagnosis of Cystic Fibrosis Through Neonatal Screening Prevents Severe Malnutrition and Improves Long-Term Growth. Wisconsin Cystic Fibrosis Neonatal Screening Study Group—Farrell PM, Kosorok MR, Rock MJ, Laxova A, Zeng L, Lai HC, et al. Pediatrics 2001 Jan;107(1):1–13.

OBJECTIVE: Despite its relative frequency among autosomal recessive diseases and the availability of the sweat test, cystic fibrosis (CF) has been difficult to diagnose in early childhood, and delays can lead to severe malnutrition, lung disease, or even death. The Wisconsin CF Neonatal Screening Project was designed as a randomized clinical trial to assess the benefits and risks of early diagnosis through screening. In addition, the incidence of CF was determined, and the validity of our randomization method assessed by comparing 16 demographic variables. METHODOLOGY: Immunoreactive trypsinogen analysis was applied to dried newborn blood specimens for recognition of CF risk from 1985 to 1991 and was coupled to DNA-based detection of the DeltaF508 mutation from 1991 to 1994. Randomization of 650 341 newborns occurred when their blood specimens reached the Wisconsin screening laboratory. This created 2 groups: an early diagnosis, screened cohort and a standard diagnosis or control group. To avoid selection bias, we devised a unique unblinding method with a surveillance program to completely identify the control subjects. Because sequential analysis of nutritional outcome measures revealed significantly better growth in screened patients during 1996, we accelerated the unblinding and completely identified the control group by April 1998. Having each member of this cohort enrolled and evaluated for at least 1 year and having completed a comprehensive surveillance program, we performed another statistical analysis of anthropometric evaluated indices that includes all CF patients without meconium ileus. RESULTS: The incidence of classical CF, ie, patients diagnosed in this trial with a sweat chloride of 60 mEq/L greater, was 1:4189. By incorporating other CF patients born during the randomization period, including 2 autopsy diagnosed patients and 8 probable patients, we calculate a maximum incidence of 1:3938 (95% confidence interval: 3402–4611). Although there were group differences in the proportion of patients with DeltaF508 genotypes and with pancreatic insufficiency, validity of the randomization plan was demonstrated by analyzing 16 demographic variables and finding no significant difference after adjustment for multiple comparisons. Focusing on patients without meconium ileus, we found a marked difference in the mean ± standard deviation age of diagnosis for screened patients (13 ± 37 weeks), compared with the standard diagnosis group (100 ± 117). Anthropometric indices of nutritional status were significantly higher at diagnosis in the screened group, including length/height, weight, and head circumference. During 13 years of study, despite similar nutritional therapy and the inherently better pancreatic status of the control group, analysis of nutritional outcomes revealed significantly greater growth associated with early diagnosis. Most impressively, the screened group had a much lower proportion of patients with weight and height data below the 10th percentile throughout childhood. CONCLUSIONS: Although the screened group had a higher proportion of patients with pancreatic insufficiency, their growth indices were significantly better than those of the control group during the 13-year follow-up evaluation and, therefore, this randomized clinical trial of early CF diagnosis must be interpreted as unequivocally positive. Our conclusions did not change when the height and weight data before 4 years of age for the controls detected by unblinding were included in the analysis. Also, comparison of growth outcomes after 4 years of age in all subjects showed persistence of the significant differences. Therefore, selection bias has been eliminated as a potential explanation. In addition, the results show that severe malnutrition persists after delayed diagnosis of CF and that catch-up may not be possible. We conclude that early diagnosis of CF through neonatal screening combined with aggressive nutritional therapy can result in significantly enhanced long-term nutritional status.

Qualitative Study of Views of Health Professionals and Patients on Guided Self Management Plans for Asthma—Jones A, Pill R, Adams S. BMJ 2000 Dec 16;321(7275):1507–1510.

OBJECTIVES: To explore the views held by general practitioners, practice nurses, and patients about the role of guided self management plans in asthma care. DESIGN: Qualitative study using nine focus groups that each met on two occasions. SETTING: South Wales. SUBJECTS: 13 asthma nurses, 11 general practitioners (six with an interest in asthma), and 32 patients (13 adults compliant with treatment, 12 non-compliant adults, and seven teenagers). RESULTS: Neither health professionals nor patients were enthusiastic about guided self management plans, and, although for different reasons, almost all participants were ambivalent about their usefulness or relevance. Most professionals opposed their use. Few patients reported sustained use, and most felt that plans were largely irrelevant to them. The attitudes associated with these views reflect the gulf between the professionals' concept of the "responsible asthma patient" and the patients' view. CONCLUSIONS: Attempts to introduce self guided management plans in primary care are unlikely to be successful. A more patient centred, patient negotiated plan is needed for asthma care in the community

DuoNeb™

(Ipratropium Bromide 0.5 mg/Albuterol Sulfate 3.0 mg*)

Inhalation Solution

***Equivalent to 2.5 mg albuterol base**

BRIEF SUMMARY OF PRESCRIBING INFORMATION

INDICATIONS AND USAGE

DuoNeb is indicated for the treatment of bronchospasm associated with COPD in patients who require more than one bronchodilator.

CONTRAINDICATIONS

DuoNeb is contraindicated in patients with a history of hypersensitivity to any of its components or to atropine and its derivatives.

WARNINGS

Paradoxical Bronchospasm. In the clinical studies of DuoNeb, paradoxical bronchospasm was not observed. However, paradoxical bronchospasm has been observed with both inhaled ipratropium bromide and albuterol products and can be life-threatening. If this occurs, DuoNeb should be discontinued immediately and alternative therapy instituted.

Do Not Exceed Recommended Dose. Fatalities have been reported in association with excessive use of inhaled products containing sympathomimetic amines and with the home use of nebulizers.

Cardiovascular Effect. DuoNeb, like other beta adrenergic agonists, can produce a clinically significant cardiovascular effect in some patients as measured by pulse rate, blood pressure, and/or symptoms. Although such effects are uncommon for DuoNeb at recommended doses, if they occur, the drug may need to be discontinued. In addition, beta agonists have been reported to produce ECG changes, such as flattening of the T wave, prolongation of the QTc interval, and ST segment depression. The clinical significance of these findings is unknown. Therefore, DuoNeb, like other sympathomimetic amines, should be used with caution in patients with cardiovascular disorders, especially coronary insufficiency, cardiac arrhythmias, and hypertension.

Immediate Hypersensitivity Reactions: Immediate hypersensitivity reactions to albuterol and/or ipratropium bromide may occur after the administration of DuoNeb, as demonstrated by rare cases of urticaria, angioedema, rash, pruritus, oropharyngeal edema, bronchospasm, and anaphylaxis.

PRECAUTIONS

General

1. Effects Seen with Sympathomimetic Drugs. As with all products containing sympathomimetic amines, DuoNeb should be used with caution in patients with cardiovascular disorders, especially coronary insufficiency, cardiac arrhythmias, and hypertension; in patients with convulsive disorders, hyperthyroidism, or diabetes mellitus; and in patients who are unusually responsive to sympathomimetic amines. Large doses of intravenous albuterol have been reported to aggravate pre-existing diabetes mellitus and ketoacidosis. Additionally, β-agonists may cause a decrease in serum potassium in some patients, possibly through intracellular shunting. The decrease is usually transient, not requiring supplementation.

2. Effects Seen with Anticholinergic Drugs. Due to the presence of ipratropium bromide in DuoNeb, it should be used with caution in patients with narrow-angle glaucoma, prostatic hypertrophy, or bladder-neck obstruction.

3. Use in Hepatic or Renal Diseases. DuoNeb has not been studied in patients with hepatic or renal insufficiency. It should be used with caution in these patient populations.

Information for Patients

The action of DuoNeb should last up to 5 hours. DuoNeb should not be used more frequently than recommended. Patients should be instructed not to increase the dose or frequency of DuoNeb without consulting their healthcare provider. If symptoms worsen, patients should be instructed to seek medical consultation.

Patients must avoid exposing their eyes to this product as temporary papillary dilation, blurred vision, eye pain, or precipitation or worsening of narrow-angle glaucoma may occur, and therefore proper nebulizer technique should be assured, particularly if a mask is used.

If a patient becomes pregnant or begins nursing while on DuoNeb, they should contact their healthcare provider about use of DuoNeb.

See the illustrated Patient's Instruction for Use in the product package insert.

Drug Interactions

Anticholinergic agents. Although ipratropium bromide is minimally absorbed into the systemic circulation, there is some potential for an additive interaction with concomitantly used anticholinergic medications. Caution is, therefore, advised in the co-administration of DuoNeb with other drugs having anticholinergic properties.

β-adrenergic agents. Caution is advised in the co-administration of DuoNeb and other sympathomimetic agents due to the increased risk of adverse cardiovascular effects.

β-receptor blocking agents. These agents and albuterol sulfate inhibit the effect of each other. β-receptor blocking agents should be used with caution in patients with hyperreactive airways, and if used, relatively selective β, selective agents are recommended.

Diuretics. The electrocardiogram (ECG) changes and/or hypokalemia that may result from the administration of non-potassium sparing diuretics (such as loop or thiazide diuretics) can be acutely worsened by β-agonists, especially when the recommended dose of the β-agonist is exceeded. Although the clinical significance of these effects is not known, caution is advised in the co-administration of β-agonist-containing drugs, such as DuoNeb, with non-potassium sparing diuretics.

Monoamine oxidase inhibitors or tricyclic antidepressants. DuoNeb should be administered with extreme caution to patients being treated with monoamine oxidase inhibitors or tricyclic antidepressants, or within 2 weeks of discontinuation of such agents because the action of albuterol sulfate on the cardiovascular system may be potentiated.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Albuterol Sulfate. In a 2-year study in Sprague-Dawley rats, albuterol sulfate caused a significant dose-related increase in the incidence of benign leiomyomas of the mesovarium at and above dietary doses of 2 mg/kg (approximately equal to the maximum recommended daily inhalation dose for adults on a mg/m² basis). In another study, this effect was blocked by the coadministration of propranolol, a non-selective beta-adrenergic antagonist.

In an 18-month study in CD-1 mice, albuterol sulfate showed no evidence of tumorigenicity at dietary doses up to 500 mg/kg (approximately 140 times the maximum recommended daily inhalation dose for adults on a mg/m² basis). In a 22-month study in Golden hamsters, albuterol sulfate showed no evidence of tumorigenicity at dietary doses up to 50 mg/kg (approximately 20 times the maximum recommended daily inhalation dose for adults on a mg/m² basis).

Albuterol sulfate was not mutagenic in the Ames test or a mutation test in yeast. Albuterol sulfate was not clastogenic in a human peripheral lymphocyte assay or in an AH1 strain mouse micronucleous assay.

Reproduction studies in rats demonstrated no evidence of impaired fertility at oral doses of albuterol sulfate up to 50 mg/kg (approximately 25 times the maximum recommended daily inhalation dose for adults on a mg/m² basis).

Ipratropium bromide. In 2-year studies in Sprague-Dawley rats and CD-1 mice, ipratropium bromide showed no evidence of tumorigenicity at oral doses up to 6 mg/kg (approximately 15 times and 8 times the maximum recommended daily inhalation dose for adults in rats and mice respectively, on a mg/m² basis).

Ipratropium bromide was not mutagenic in the Ames test and mouse dominant lethal test. Ipratropium bromide was not clastogenic in a mouse micronucleous assay.

A reproduction study in rats demonstrated decreased conception and increased resorptions when ipratropium bromide was administered orally at a dose of 90 mg/kg (approximately 240 times the maximum recommended daily inhalation dose for adults on a mg/m² basis). These effects were not seen with a dose of 50 mg/kg (approximately 140 times the maximum recommended daily inhalation dose for adults on a mg/m² basis).

Pregnancy

TERATOGENIC EFFECTS. Pregnancy Category C

Albuterol Sulfate. *Pregnancy Category C.* Albuterol sulfate has been shown to be teratogenic in mice. A study in CD-1 mice given albuterol sulfate subcutaneously showed cleft palate formation in 5 of 111 (4.5%) fetuses at 0.25 mg/kg (less than the maximum recommended daily inhalation dose for adults on a mg/m² basis) and in 10 of 108 (9.3%) fetuses at 2.5 mg/kg (approximately equal to the maximum recommended daily inhalation dose for adults on a mg/m² basis). There are no adequate and well-controlled studies in pregnant women. DuoNeb should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

A study in rabbits revealed [illegible] at a dose of 10 mg/kg (approximately 55 times the maximum recommended daily inhalation dose for adults on a mg/m² basis).

A study in which pregnant rats were dosed with [illegible] maternally derived from the maternal circulation by the fetus.

There are no adequate and well-controlled studies in pregnant women. [illegible] should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Ipratropium bromide. *Pregnancy Category B.* Reproduction studies in CD-1 mice, Sprague-Dawley rats, and New Zealand rabbits demonstrated no evidence of teratogenicity at oral doses up to 10, 100, and 125 mg/kg, respectively (approximately 15, 270, and 680 times the maximum recommended daily inhalation dose for adults on a mg/m² basis). Reproduction studies in rats and rabbits demonstrated no evidence of teratogenicity at inhalation doses up to 1.5 and 1.8 mg/kg, respectively (approximately 4 and 10 times the maximum recommended daily inhalation dose for adults on a mg/m² basis). There are no adequate and well-controlled studies of the use of DuoNeb, albuterol sulfate, or ipratropium bromide in pregnant women. DuoNeb should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Labor and Delivery

Oral albuterol sulfate has been shown to delay preterm labor in some reports. Because of the potential of albuterol to interfere with uterine contractility, use of DuoNeb during labor should be restricted to those patients in whom the benefits clearly outweigh the risks.

Nursing Mothers

It is not known whether the components of DuoNeb are excreted in human milk. Although ipratropium bromide, a quaternary bases pass into breast milk, it is unlikely that ipratropium bromide would reach the infant in an important extent, especially when taken as a nebulized solution. Because of the potential for tumorigenicity shown for albuterol sulfate in some animals, a decision should be made whether to discontinue nursing or discontinue DuoNeb, taking into account the importance of the drug to the mother.

Pediatric Use

The safety and effectiveness of DuoNeb in patients below 18 years of age have not been established.

Geriatric Use

Of the total number of subjects in clinical studies of DuoNeb, 62 percent were 65 and over, while 19 percent were 75 and over. No overall differences in safety or effectiveness were observed between these subjects and younger subjects, and other reported clinical experience has not identified differences in responses between the elderly and younger patients, but greater sensitivity of some older individuals cannot be ruled out.

ADVERSE REACTIONS

Adverse reaction information concerning DuoNeb was derived from the 12-week controlled clinical trial.

ADVERSE EVENTS OCCURRING IN ≥ 1% OF ≥ 1 TREATMENT GROUP(S) AND WHERE THE COMBINATION TREATMENT SHOWED THE HIGHEST PERCENTAGE

Body System COSTART Term	Albuterol n (%)	Ipratropium n (%)	DuoNeb n (%)
NUMBER OF PATIENTS	761	754	765
N (%) Patients with AE	327 (43.0)	329 (43.6)	367 (48.0)
BODY AS A WHOLE			
Pain	8 (1.1)	4 (0.5)	10 (1.3)
Pain chest	11 (1.4)	14 (1.9)	20 (2.6)
DIGESTIVE			
Diarrhea	5 (0.7)	9 (1.2)	14 (1.8)
Dyspepsia	7 (0.9)	8 (1.1)	10 (1.3)
Nausea	7 (0.9)	6 (0.8)	11 (1.4)
MUSCULO-SKELETAL			
Cramps leg	8 (1.1)	6 (0.8)	11 (1.4)
RESPIRATORY			
Bronchitis	11 (1.4)	13 (1.7)	13 (1.7)
Lung Disease	36 (4.7)	34 (4.5)	49 (6.4)
Pharyngitis	27 (3.5)	27 (3.6)	34 (4.4)
Pneumonia	7 (0.9)	8 (1.1)	10 (1.3)
UROGENITAL			
Infection urinary tract	3 (0.4)	9 (1.2)	12 (1.6)

Additional adverse reactions reported in more than 1% of patients treated with DuoNeb included constipation and voice alterations.

In the clinical trial, there was a 0.3% incidence of possible allergic-type reactions, including skin rash, pruritus, and urticaria.

Additional information derived from the published literature on the use of albuterol sulfate and ipratropium bromide singly or in combination includes precipitation or worsening of narrow-angle glaucoma, acute eye pain, blurred vision, paradoxical bronchospasm, wheezing, exacerbation of COPD symptoms, drowsiness, aching, flushing, upper respiratory tract infection, palpitations, taste perversion, elevated heart rate, sinusitis, back pain, and sore throat.

DOSAGE AND ADMINISTRATION

The recommended dose of DuoNeb is one 3 mL vial administered 4 times per day via nebulization with up to 2 additional 3 mL doses allowed per day, if needed. Safety and efficacy of additional doses or increased frequency of administration of DuoNeb beyond these guidelines has not been studied and the safety and efficacy of extra doses of albuterol sulfate or ipratropium bromide in addition to the recommended doses of DuoNeb have not been studied.

The use of DuoNeb can be continued as medically indicated to control recurring bouts of bronchospasm. If a previously effective regimen fails to provide the usual relief, medical advice should be sought immediately, as this is often a sign of worsening COPD, which would require reassessment of therapy.

A Pan-LC-Plus™ nebulizer (with face mask or mouthpiece) connected to a PRONEB™ compressor was used to deliver DuoNeb to each patient in one U.S. clinical study. The safety and efficacy of DuoNeb delivered by other nebulizers and compressors have not been established.

DuoNeb should be administered via jet nebulizer connected to an air compressor with an adequate air flow, equipped with a mouthpiece or suitable face mask.

HOW SUPPLIED

DuoNeb is supplied as a 3-mL sterile solution for nebulization in sterile low-density polyethylene unit-dose vials. Cards of five vials are placed into a foil pouch. Supplied in cartons as listed below:

NDC 49502-672-30	30 vials per carton
NDC 49502-672-60	60 vials per carton

Store between 2°C and 25°C (36°F and 77°F). Protect from light.

DEY
DEY Napa, CA 94558

03-485-21(BRS) May 2001

1. Gross N, Tashkin D, Miller R, et al. Inhalation by nebulization of albuterol-ipratropium combination (Dey combination) is superior to either agent alone in the treatment of chronic obstructive pulmonary disease. *Respiration.* 1998;65:354-362.

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It's the first and only one-step dual-therapy nebulizer solution for COPD, with improved efficacy over albuterol or ipratropium alone—and no increased safety risk.[1]

DuoNeb is indicated for the treatment of bronchospasm associated with COPD in patients requiring more than one bronchodilator. In a 12-week clinical study, the most common adverse reactions reported with DuoNeb Inhalation Solution were chest pain, pharyngitis, diarrhea, bronchitis, nausea and leg cramps.

Albuterol sulfate can have a significant cardiovascular effect, significant hypokalemia and the potential life-threatening paradoxical bronchospasm. Caution is advised in patients with convulsive disorders, hyperthyroidism, diabetes mellitus, narrow-angle glaucoma, prostatic hypertrophy or bladder-neck obstruction. Co-administration of DuoNeb Inhalation Solution and other sympathomimetic agents may increase the risk of adverse cardiovascular effects.

Please see following page for prescribing information.

DuoNeb™
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Inhalation Solution
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The first and only one-step dual-therapy nebulizer solution for COPD.

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3.0 mg Albuterol Sulfate
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Acute Exacerbation of Idiopathic Pulmonary Fibrosis and Fibroproliferative Acute Respiratory Distress Syndrome: Similar Disease, Similar Treatment?

Idiopathic pulmonary fibrosis (IPF), also referred to as cryptogenic fibrosing alveolitis, is a progressive interstitial lung disease of unknown etiology. Usual interstitial pneumonitis (UIP) is pathologically the most common idiopathic interstitial pneumonia producing the clinical entity of IPF.[1] IPF is a chronic condition, characterized by the insidious onset of dyspnea and cough over several months. IPF tends to be a slowly progressive disease, with mean survival times ranging from 15 months to 5.6 years.[1]

See the Case Study on Page 698

Although described in only a few reports in the literature, some IPF patients may develop an acute decompensation, causing fulminant respiratory failure. This abrupt decompensation has been referred to as acute exacerbation of IPF and as accelerated IPF. Kondoh et al[2] described 3 IPF patients who developed acute exacerbations of IPF, defined as: (1) exacerbation of dyspnea within a few weeks, (2) newly developing diffuse infiltrates on chest radiograph, (3) deterioration of oxygenation, with the ratio of arterial partial pressure of oxygen to fraction of inspired oxygen being less than 225 mm Hg, and (4) absence of apparent infectious agents. The diagnosis of IPF was made 6 months to 2 years prior to the acute deterioration. Initial symptoms of the acute exacerbation began with an influenza-like illness or cough with fever for 3–20 days prior to admission. Open lung biopsies from the 3 patients were consistent with acute lung injury pattern superimposed on UIP. Cultures of bronchoalveolar lavage (BAL) fluid and biopsy specimens and serum titers for various viruses showed no evidence of an infectious process causing the deterioration. All 3 patients improved with corticosteroid therapy, although 1 patient died 5 months later because of *Staphylococcus aureus* infection.

Using high-resolution computed tomography, Akira et al evaluated 17 IPF patients who developed aecelerated deterioration.[3] The duration of symptoms before deterioration ranged from 5 months to 10 years. High-resolution computed tomography showed peripheral infiltrates in 6 patients, multifocal infiltrates in 6 patients, and diffuse infiltrates in 5 patients. Biopsy or autopsy specimens showed acute diffuse alveolar damage (DAD) in conjunction with UIP in the patients with multifocal and diffuse infiltrates, whereas acute fibroblastic foci were noted in the patients with peripheral infiltrates. Despite corticosteroid treatment, all 5 patients with diffuse infiltrates died, whereas 3 of 6 patients with multifocal infiltrates died and 1 of 6 patients with peripheral infiltrates died. Similar findings with diffuse alveolar damage superimposed on UIP have been noted by other authors.[4]

In this issue of Respiratory Care, Nishiyama et al describe a 74 year-old man with UIP who developed an acute exacerbation of IPF with respiratory failure requiring mechanical ventilation.[5] The patient improved with a prolonged course of corticosteroid therapy, using a regimen published by Meduri et al to treat unresolving acute respiratory distress syndrome (ARDS).[6] Infection as a cause of the patient's decompensation was excluded by BAL cultures and negative serologies for viruses *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, and *Legionella pneumophila*. Though the patient's clinical course is consistent with the few published series of patients with acute exacerbation of IPF, tissue biopsy during the patient's acute decompensation was not performed to confirm this diagnosis and exclude other potential etiologies for the observed deterioration.

With few reports describing acute exacerbations of IPF in the literature, the optimal treatment for this entity is unclear. Corticosteroids are used by many clinicians, although there are little data to make recommendations for dose and duration of therapy. As previously mentioned, Kondoh et al[2] successfully treated 3 patients with 1,000 mg/d of methylprednisolone for 3 days, followed by a taper based on patient response. Akira et al[3] successfully treated 9 of 17 patients with 1,000 mg of methylprednisolone, although the duration of treatment at that dose and taper were not specified.

In their discussion, Nishiyama et al note that patients with acute exacerbation of IPF have organizing DAD as the main histologic feature in addition to the findings of UIP. Given that the histology of late-phase ARDS also demonstrates organizing DAD, Nishiyama et al hypothesize that treatments used for ARDS may benefit patients with acute IPF exacerbations, and this was their rationale for prolonged corticosteroid therapy, using the regimen previously reported by Meduri et al[6] to treat patients with unresolving ARDS. Other than comparable histopathologic features, it is unclear if there are any other similarities between ARDS and acute exacerbation of IPF in terms of

pathophysiology and cytokine response. DAD has many causes, including infection, sepsis, shock, trauma, noxious gas exposure, drug reactions, and collagen vascular diseases.[4] Idiopathic DAD has also been described in patients with acute interstitial pneumonitis, considered synonymous with Hamman-Rich syndrome by many authors.[7,8] Acute interstitial pneumonitis has clinical findings similar to acute exacerbation of IPF, but patients do not have preexisting lung disease and, if they survive, patients do not progress to IPF.

Fibroproliferation is a stereotypical reparative response to lung injury and occurs in the repair phase of DAD and within 7 days of the onset of ARDS, with a rapid increase in the second and third weeks of continued ARDS. Fibroproliferation is characterized by the replacement of damaged epithelial cells by accumulation of mesenchymal cells such as fibroblasts and their connective tissue products such as collagen in the airspaces. Extensive fibrotic remodeling of the lung parenchyma from unchecked fibroproliferation has been noted in ARDS patients. There is evidence that corticosteroids may be beneficial in ARDS during this fibroproliferative phase.[9]

Treatment with corticosteroids has been shown in one study to reduce plasma and BAL concentrations of tumor necrosis factor α, interleukin 1β, interleukin 6, and interleukin 8. Higher initial levels and more persistently elevated levels of those cytokines have been observed in nonsurvivors of ARDS than in survivors.[9,10] Decreases in plasma and BAL procollagen amino-terminal propeptide type I and type III, which are secreted by fibroblasts and reflect collagen synthesis, with accompanying improvements in oxygenation, have been reported in one study of ARDS patients treated with corticosteroids.[11]

In the randomized trial by Meduri et al comparing corticosteroid treatment for 32 days to placebo in 24 patients with unresolving ARDS, overall survival was 87% (14 of 16) in the corticosteroid treatment group versus 37% (3 of 8) in the placebo group.[6] Enrollment was stopped after 24 patients based on interim statistical analysis. Other (uncontrolled) case series have also reported benefit from corticosteroids in treating fibroproliferative or unresolving ARDS.[9]

With a similar process of active fibroblastic proliferation being observed in biopsy specimens from patients with acute exacerbation of IPF, there is some rationale for corticosteroid treatment for this entity. Given the life-threatening nature of acute exacerbation of IPF and positive responses to corticosteroids being noted in the literature, using corticosteroids to treat these exacerbations appears appropriate, although in general corticosteroids are beneficial in the minority of UIP patients. Duration of therapy may or may not be important. In experimental acute lung injury, corticosteroid administration was effective in decreasing edema and lung collagen formation with prolonged treatment, although steroid withdrawal rapidly negated this beneficial effect.[12–14] With the paucity of published series in the literature, it is difficult to make recommendations regarding dose and duration of therapy. In the absence of other data, however, it seems reasonable to use the regimen of prolonged low-dose corticosteroid therapy described by Meduri et al for fibroproliferative ARDS, although a relationship between ARDS and acute exacerbation of IPF has yet to be established. Additional studies of this condition would be most welcome.

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Evaluation of Inspiratory Rise Time and Inspiration Termination Criteria in New-Generation Mechanical Ventilators: A Lung Model Study

Sunisa Chatmongkolchart MD, Purris Williams RRT, Dean R Hess PhD RRT FAARC, and Robert M Kacmarek PhD RRT FAARC

INTRODUCTION: Inspiratory rise time adjustment during pressure ventilation and inspiration termination criteria adjustment during pressure support ventilation are available on some of the newest mechanical ventilators. Both are designed to improve patient-ventilator synchrony. However, the function of these adjuncts during pressure ventilation on these ventilators has not been evaluated. METHODS: Three inspiratory rise times (minimum, medium, and maximum) were evaluated in 5 new-generation mechanical ventilators (Hamilton Galileo, Siemens 300A, Puritan Bennett 840, Bear 1000, and Dräger Evita 4) during pressure support and pressure assist/control. Three inspiration termination criteria settings (minimum, medium, and maximum) were also evaluated in 2 mechanical ventilators (Hamilton Galileo and Puritan Bennett 840) during pressure support. All evaluations were performed with a spontaneous breathing lung model (compliance 50 mL/cm H_2O, resistance 8.2 cm H_2O/L/s, respiratory rate 12 breaths/min, inspiratory time 1.0 s, and lung model peak inspiratory flow 60 L/min). Throughout the evaluation, inspiratory pressure was set at 15 cm H_2O and positive end-expiratory pressure at 5 cm H_2O, resulting in a peak airway pressure of 20 cm H_2O. RESULTS: Significant ($p < 0.05$) and important (> 10%) differences were found among the ventilators at similar rise times (minimum, medium, and maximum) and for each ventilator as rise time was varied. Also, significant ($p < 0.05$) and important (> 10%) differences were observed between ventilators and within each ventilator when inspiration termination criteria were varied. There were significant ($p < 0.05$) differences between pressure support and pressure assist/control, but most were < 10%, except those associated with expiration. CONCLUSIONS: Major differences exist for each ventilator as rise time or inspiration termination criteria are varied and among ventilators at similar settings. Inspiration termination criteria adjustment markedly affects transition to exhalation in the Puritan Bennett 840. *Key words: mechanical ventilation, inspiratory rise time, inspiration termination criteria, pressure support, pressure assist/control, flow-triggered, flow-cycled.* [Respir Care 2001;46(7):666–677]]

Introduction

During patient-triggered mechanical ventilatory assistance, patient-ventilator synchrony is a major problem. Many investigators have demonstrated that volume ventilation commonly results in large patient efforts during patient-triggered breaths, because of fixed inspiratory flow patterns.[1,2] As a result, pressure ventilation (pressure support [PS] and pressure assist/control [PA/C]) has been recommended as the optimal approach to ensure patient-ventilator synchrony, because inspiratory flow delivery responds to patient demand.[3,4] However, as nicely illustrated by MacIntyre et al[5] and others,[6,7] dyssynchrony can also exist during pressure ventilation. If the rise in flow is not commensurate with patient demand, pressure may overshoot the set level (too rapid an increase in flow) or patient effort can be excessive (inadequate increase in flow). Both

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of these extremes can result in patient-ventilatory dyssynchrony. A number of the newest generation of mechanical ventilators give the clinician the option of setting the rise time during pressure ventilation to meet patient demand.

During PS, inspiration normally cycles to expiration when inspiratory flow decreases to a level equal to a fixed percentage (eg, 25%) of the peak flow or to a specified flow (eg, 5 L/min).[8] However, if the patient decides to terminate inspiration before the flow criteria are met, the patient must activate abdominal muscles to force airway pressure to rise to a fixed level above the set pressure target (eg, 3 cm H_2O).[9] This activation of the expiration muscles at the end of the inspiratory phase increases patient effort and produces dyssynchrony.[5] A few of the newest generation of ventilators have the ability to adjust the inspiration termination criteria.

Limited information is currently available on the function of rise time and inspiration termination criteria on the newest generation of ventilators. We evaluated the function of rise time on 5 new-generation ventilators and inspiration termination criteria on 2 new-generation ventilators. We hypothesized that there would be no differences in the operation of rise time between PS and PA/C modes on a given ventilator but that there would be large differences among ventilators evaluated. We also hypothesized that a synchronous transition to exhalation would be made possible by appropriate adjustment of the inspiration termination criteria.

Methods

Lung Model

All evaluations were performed with a simulated spontaneous breathing lung model, as previously described.[10,11] The lung model consisted of 2 parallel bellows suspended in an airtight rigid box (Fig. 1). The space in the rigid box represents the pleural space, which was connected to a low-compliance T tube through which gas flow was injected to create a negative pleural pressure (P_{pl}). Source gas (oxygen, 50 psi) was connected to an air pressure regulator (SMC air pressure regulator model AR 2000, SMC Company, Tokyo, Japan) and a proportional solenoid valve (SMC 315, SMC Company, Tokyo, Japan). Opening of the solenoid valve was controlled by a function generator (EGC 2230, Kenwood, Tokyo, Japan). Operation of the regulator allowed adjustment of negative pressure in the pleural space and simulation of a spontaneous breathing pattern. Thus, inspiratory flow demand, respiratory rate, and inspiratory time were controlled independently. A linear resister (7100 R-5, Hans Rudolph, Kansas City, Missouri) established a resistance of 8.2 cm H_2O/L/s at a flow of 60 L/min. Compliance of 50 mL/cm H_2O was set by springs on the bellows. The lung model

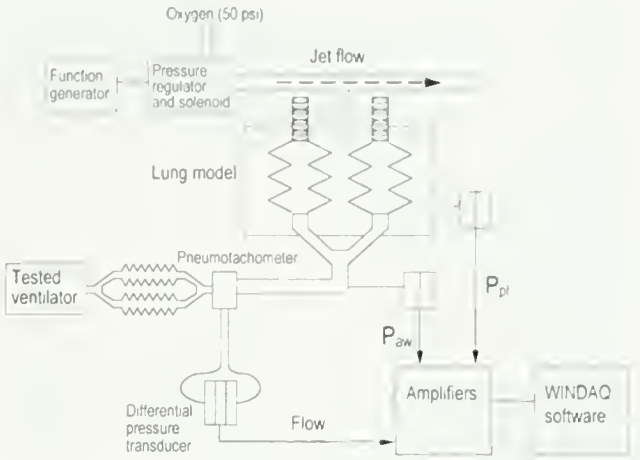

Fig. 1. Experimental set-up. See text for details. P_{aw} = airway pressure, P_{pl} = pleural pressure.

was connected to each studied ventilator using a standard ventilator circuit. A humidifier (ConchaTherm IV heated humidifier, Hudson RCI, Temecula, California) was maintained in the circuit, but active humidification was not employed.

Three inspiratory rise times (minimum, medium, and maximum) (Table 1) were studied on 5 ventilators, the Hamilton Galileo (Hamilton Medical AG, Rhazuns, Switzerland), the Siemens 300A (Siemens-Elema, Solna, Sweden), the Puritan Bennett 840 (Puritan Bennett, Pleasanton, California), the Bear 1000 (ThermoRespiratory Group, Palm Springs, California), and the Dräger Evita 4 (Dräger, Telford, Pennsylvania) during both PS and PA/C modes. The evaluations were performed at ventilator settings of inspiratory pressure of 15 cm H_2O and positive end-expiratory pressure (PEEP) of 5 cm H_2O. During PA/C mode the inspiratory time was set at 1.0 s. The lung model was always set at a respiratory rate of 12 breaths/min, inspiratory time of 1.0 s, and peak inspiratory flow of 60 L/min. The Hamilton Galileo and Puritan Bennett 840 (PB 840) were also evaluated at 3 inspiration termination criteria settings (minimum, medium, and maximum) (Table 2) in PS mode. After a period of stabilization, flow, airway pressure, and pleural pressure were recorded for a series of breaths at each experimental condition. Three consecutive breaths for each experimental setting were evaluated (Fig. 2). The inspiration termination criteria of every studied ventilator except the Galileo and the PB 840 were automatically fixed at predetermined levels by the manufacturers. The inspiration termination criteria of the Galileo and the PB 840 were set at 25% during assessment of inspiratory rise times. When the effect of the inspiration termination criteria was evaluated, the inspiratory rise time was set at the maximum: 50 ms for the Hamilton Galileo and 100% for the PB 840. All ventilators were set to flow-triggering at the most sensitive level that did not

Table 1. Experimental Settings of Inspiratory Rise Time with Manufacturer-Recommended Settings

Ventilator	Minimum	Medium	Maximum	Manufacturer-recommended
Hamilton Galileo (pressure ramp, ms)	200	125	50	50
Siemens 300A (%)	10	5	0	1
PB 840 (flow acceleration, %)	1	50	100	50
BEAR 1000 (pressure slope)	9	0	9	0
Dräger Evita 4 (s)	2	1	0	0.2

PB = Puritan Bennett

result in auto-triggering. This resulted in the following settings. The Galileo inspiratory sensitivity was set at 2 L/min. The inspiratory sensitivity of the Siemens 300A was set between the green and red line (2 L/min). For the PB 840, the inspiratory sensitivity was set at 0.5 L/min, with base flow of 2 L/min. The sensitivity of the BEAR 1000 was set at 1 L/min, with bias flow of 5 L/min. For the Dräger Evita 4, the sensitivity was set at 0.3 L/min.

Measurement and Calibration

Flow was measured at the distal end of the breathing circuit, using a pneumotachometer (model 3700 A, Hans Rudolph Inc, Kansas City, Missouri) connected to a differential pressure transducer (MP 45–14-871, ± 2 cm H_2O, Validyne, Northridge, California). The pneumotachometer was calibrated with a 1 L/s flow delivered by a precision flow meter (Brooks Instruments, Hatfield, Pennsylvania). Pressures at the airway opening at the proximal end of the endotracheal tube and in the simulated pleural space (P_{pl}) were measured with differential pressure transducers (MP 45–32-871, ± 100 cm H_2O, Validyne, Northridge, California). The pressure transducers were calibrated at 20 cm H_2O with a water manometer. All signals were amplified (model 8805C, Hewlett Packard, Waltham, Massachusetts), sampled at 100 Hz, digitized, and recorded with WINDAQ software (Dataq Instruments, Akron, Ohio). The WINDAQ playback system was used to analyze the data.

Studied Variables

Variables studied are graphically illustrated in Figure 2. The beginning and end of inspiration were identified based on changes in the simulated pleural pressure. A below-baseline deflection in pleural pressure identified the start of inspiration by the lung model. The onset of the return of pleural pressure toward baseline identified the termination of inspiration by the lung model. These pleural pressure changes were used in determining the duration of inspiration and expiration.

Table 2. Experimental Settings of Inspiratory Termination Criteria and Manufacturer-Recommended Settings

Ventilator	Minimum	Medium	Maximum	Manufacturer-recommended
Hamilton Galileo (%)	40	25	10	25
PB 840 (%)	45	25	1	10

PB = Puritan Bennett

The following variables were measured during the inspiratory phase:

1. Inspiratory delay time. The time from the onset of inspiration until the return of the airway pressure to baseline. This represents the total delay from onset of lung model inspiration to the beginning of system pressurization. Ideally, the inspiratory delay time should be zero.

2. Inspiratory trigger time. The time from the onset of inspiration until the maximum sub-baseline trigger pressure is established. This represents the delay between initiation of inspiration and the ventilator responding with sufficient flow to stop the decline in airway pressure, indicating the responsiveness of the ventilator to patient effort. Ideally, inspiratory trigger time should be zero.

3. Time to return trigger pressure to baseline. The time from the maximum sub-baseline trigger pressure to the return of airway pressure to baseline. This represents the speed with which the ventilator pressurizes the system after patient effort is recognized and illustrates the speed with which initial flow is delivered. Ideally, the time to return trigger pressure to baseline should be zero.

4. Inspiratory trigger pressure. The pressure change from baseline to maximum sub-baseline trigger pressure. This pressure represents the pressure deflection below baseline necessary to activate the breath. Ideally, the inspiratory trigger pressure should be less than 1.0 cm H_2O.

5. Trigger-pressure time product. The area defined by the inspiratory delay time and inspiratory trigger pressure, illustrated as Area 1 in Figure 2. The trigger-pressure time product represents the amount of patient effort expended in activating a breath. Ideally, the trigger-pressure time product should be zero.

6. Inspiratory positive pressure area. The area defined by the inspiratory pressure curve beginning with the return of pressure to baseline (after inspiratory delay time) and

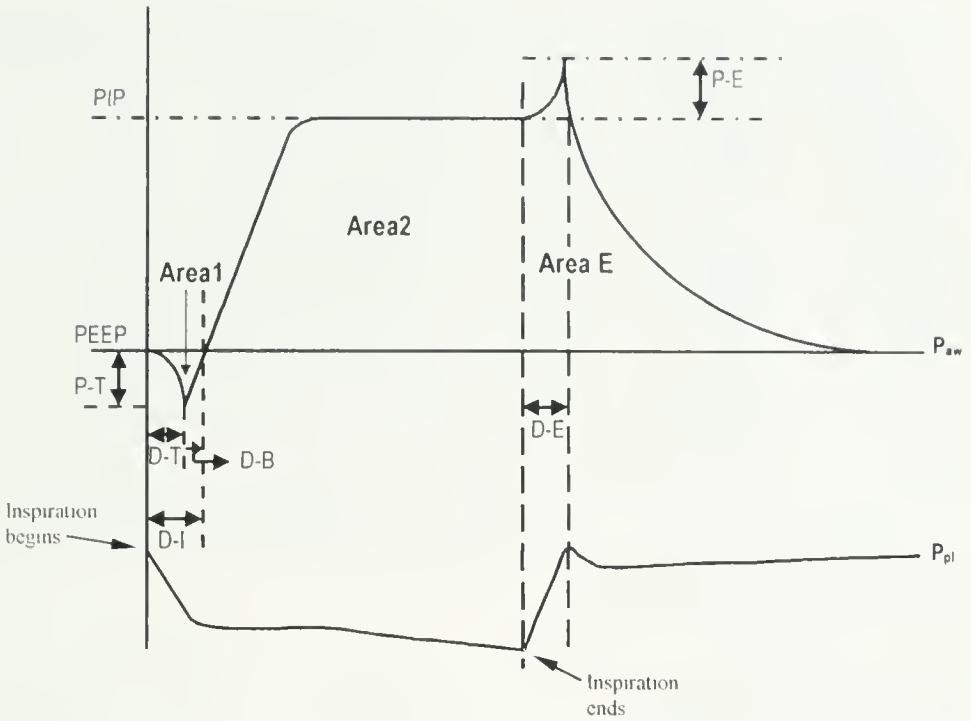

Fig. 2. Schematic illustrating the variables evaluated. See text for definitions of variables. P_{aw} = peak airway pressure. P_{pl} = pleural pressure. PIP = peak inspiratory pressure. PEEP = positive end-expiratory pressure.

ending at the time pleural pressure indicated the onset of expiration (Area 2 in Fig. 2). Area 2 represents the ability of the ventilator to pressurize the system: the actual area of pressure-versus-time applied during each inspiration.

7. Inspiratory area percent. The percentage that the remainder of Area 2 minus Area 1 represents of the area of an ideal airway pressure curve, not shown in Fig. 2, calculated as:

$$\text{Area 1\%} = [\text{Area 2} - \text{Area 1}]/[(\text{PIP} - \text{PEEP}) \times (\text{Inspiratory time})] \times 100 \quad (1)$$

wherein PIP is peak inspiratory pressure. The ideal airway pressure curve is the rectangle defined by the onset of inspiration (pleural pressure change) vertically to peak pressure extending to the time inspiration ends (pleural pressure change). This calculation is based on our assumption that the most ideal positive pressure breath would immediately pressurize the system on initiation of inspiration and immediately return to baseline at the end of inspiration. Thus, no Area 1 should be present. Optimal performance would be an Area 1% of 100%. That is, the closer the ventilation is to that level, the more optimal is its operation, under our assumptions.

8. Peak flow. The maximum ventilator-delivered flow (in L/min) obtained during the inspiratory phase.

The following expiratory variables were measured:

1. Expiratory time delay. The time between the onset of exhalation and the moment when airway pressure falls below the end-inspiratory value. This represents the amount of time it takes for the airway pressure to decrease below set inspiratory level after the lung model begins to expire. Ideally, the expiratory time delay should be zero.

2. Supra-plateau expiratory pressure change. The difference between the maximum pressure during exhalation and the end-inspiratory pressure. This represents the amount of pressure above set inspiratory level that was obtained during the transition to exhalation. Whenever this is present, it indicates that the lung model began to expire before the ventilator started exhalation. Ideally, the supra-plateau expiratory pressure change should be zero.

3. Expiratory pressure area (Area E). The area of the airway pressure curve during expiration, calculated as:

$$\text{Area E} = \int (P_{aw} - \text{PEEP})\, dt \text{ (during expiration)} \quad (2)$$

This area represents both the ability of the ventilator to coordinate with the lung model the transition to exhalation and the overall resistance of the expiratory system. Ideally, Area E should be zero.

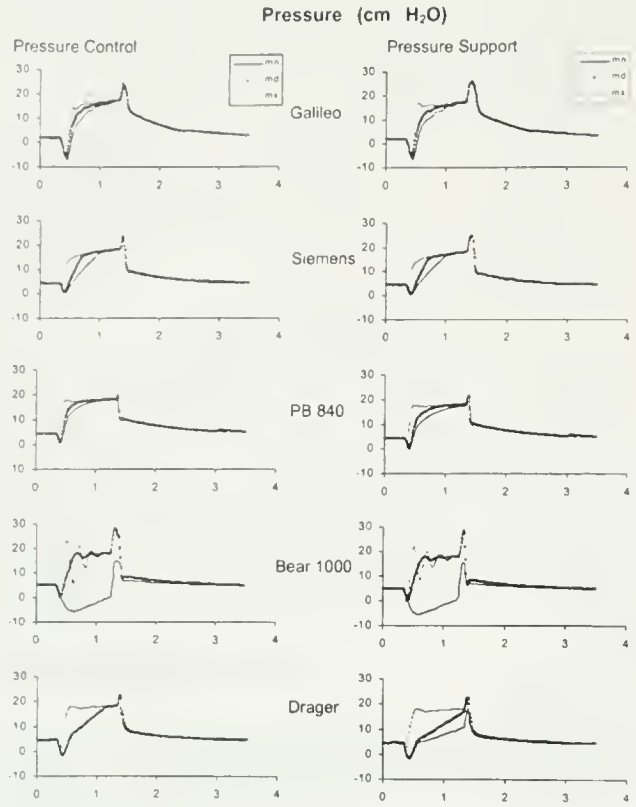

Fig. 3. Airway-pressure-versus-time waveforms for studied ventilators at minimum (mn, continuous line), medium (md, dotted line), and maximum (mx, dashed line) inspiratory rise times in pressure assist/control mode (left column) and pressure support mode (right column). With the BEAR 1000 note the rate of pressurization overshoot of the set level at maximum inspiratory rise time, whereas the rate of pressurization was unable to reach the preset pressure at the minimum inspiratory rise time. PB 840 = Puritan Bennett 840 ventilator.

Statistics

Data are expressed as mean ± standard deviation. Comparisons were done using analysis of variance. When significant differences were observed, post hoc analysis was performed with the Scheffé test. Differences were considered significant when $p < 0.05$. Because the lung model delivers very consistent breaths and, as a result, small standard deviations, very small differences (frequently within measurement error) were statistically significant. We did not consider differences clinically important unless they were more than 10%. All statistical analysis was performed using commercially available software (SPSS 9.0, SPSS, Chicago, Illinois).

Results

Inspiratory Rise Time

Comparison of Pressure Support and Pressure Assist/Control. Figures 3 and 4 present airway-pressure-versus-time and flow-versus-time waveforms for each ventilator evaluated, at all 3 rise time settings. There were significant ($p < 0.05$) differences between PS and PA/C modes during inspiration, but few were considered important (> 10% difference). Specifically, time to return trigger pressure to baseline, inspiratory delay time, trigger-pressure time product, and Area 1% differed > 10% between PS and PA/C with the Dräger at the minimum rise time setting (Tables 3 and 4, Figures 5 and 6), and Area 1% with the Dräger differed > 10% between PS and PA/C at the medium rise time setting. Trigger-pressure time product differed > 10% between PS and PA/C on the Galileo at minimum, medium, and maximum, and on the PB 840 at minimum rise time settings. However, significant and important differences were noted during expiration. At most rise time settings, Area E, supra-plateau expiratory pressure change, and expiratory time delay were greater

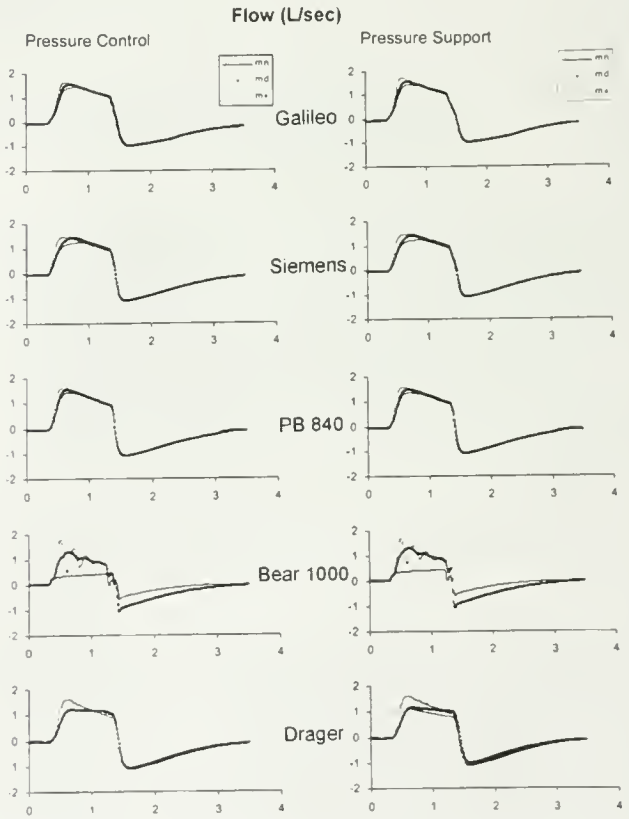

Fig. 4. Flow-versus-time waveforms of studied ventilators at minimum (mn, continuous line), medium (md, dotted line), and maximum (mx, dashed line) inspiratory rise time in pressure assist/control mode (left column) and pressure support mode (right column). With the BEAR 1000 note the rate of pressurization overshoot of the set level at maximum inspiratory rise time, whereas the rate of pressurization was unable to reach the preset pressure at minimum inspiratory rise time. PB 840 = Puritan Bennett 840 ventilator.

Table 3. Inspiratory Trigger Variables and Area E During Pressure Support at 3 Rise Time Settings

	PS	Galileo	Siemens	PB 840	Bear 1000*	Dräger
P T (cm H_2O)	minimum†	8.42 ± 0.10*§	4.16 ± 0.10§	4.59 ± 0.17§		6.61 ± 0.09‡§
	medium†	7.54 ± 0.00§	3.83 ± 0.20§	3.77 ± 0.17§	4.78 ± 0.20	6.28 ± 0.09‡
	maximum†	5.69 ± 0.34‖	1.91 ± 0.09‖	1.86 ± 0.25‖	4.43 ± 0.20	4.26 ± 0.00‖
D-T (s)	minimum†	0.12 ± 0.01‡§	0.11 ± 0.01§	0.09 ± 0.01§	—	0.12 ± 0.00‡§
	medium†	0.11 ± 0.00§	0.10 ± 0.01§	0.08 ± 0.01§	0.08 ± 0.01	0.11 ± 0.00§
	maximum†	0.09 ± 0.01‡‖	0.05 ± 0.01‡‖	0.06 ± 0.01‡‖	0.07 ± 0.00	0.07 ± 0.01‖
D-B (s)	minimum†	0.09 ± 0.00‡§	0.11 ± 0.00‡§	0.07 ± 0.01‡§		0.16 ± 0.01‡§
	medium†	0.07 ± 0.00§	0.07 ± 0.01§	0.05 ± 0.01§	0.08 ± 0.01§	0.13 ± 0.01§
	maximum†	0.05 ± 0.01‡‖	0.02 ± 0.00‡‖	0.02 ± 0.01‡‖	0.04 ± 0.00†‖	0.03 ± 0.01‖
D-I (s)	minimum†	0.21 ± 0.01‡§	0.22 ± 0.01‡§	0.16 ± 0.01‡§		0.28 ± 0.01‡§
	medium†	0.18 ± 0.00§‖	0.17 ± 0.00§‖	0.13 ± 0.00§‖	0.15 ± 0.01§	0.24 ± 0.01§‖
	maximum†	0.13 ± 0.01‡‖	0.07 ± 0.01‡‖	0.08 ± 0.00‡‖	0.11 ± 0.00‡‖	0.09 ± 0.01‡‖
Area E (cm H_2O · s)	minimum†	12.61 ± 0.14	5.87 ± 0.26	6.19 ± 0.04§	—	2.95 ± 0.18‡§
	medium†	12.58 ± 0.11	5.88 ± 0.30	6.27 ± 0.07§	4.59 ± 0.06§	4.58 ± 0.40
	maximum†	12.56 ± 0.06	6.07 ± 0.10	6.51 ± 0.05‡‖	4.24 ± 0.18§‖	4.59 ± 0.06‖

PS = pressure support
PB = Puritan Bennett
*No data available for Bear 1000 at minimum setting
P T = trigger pressure
†$p < 0.05$ among ventilators
‡$p < 0.05$ vs medium for specific ventilator
§$p < 0.05$ vs maximum for specific ventilator
‖$p < 0.05$ vs minimum for specific ventilator
D-T = inspiratory trigger time
D-B = time to return trigger pressure to baseline
D-I = inspiratory delay time
Area E = area of the airway pressure curve during exhalation

($p < 0.05$ and > 10% difference) during PS (Tables 3 and 4, Figures 7 through 9) than during PA/C.

Comparison of Rise Times (minimum vs medium vs maximum). There were significant ($p < 0.001$) and important (> 10%) differences in inspiratory trigger pressure, inspiratory trigger time, time to return trigger pressure to baseline, inspiratory delay time, and trigger-pressure time product in all ventilators, but only minor differences in Area E, supra-plateau expiratory pressure change, and expiratory time delay ($p < 0.05$) on some ventilators during both PS and PA/C as rise time was altered (Tables 3 and 4, Figs. 5–9). Peak flow varied ($p < 0.05$) in all ventilators. With the Bear and Dräger ventilators, peak flow changed > 10% as rise time varied. At the minimum rise time, only peak flow of the Bear 1000 could be derived from the waveform, increasing 66% when rise time was changed to medium. As noted in Figure 3, at the minimum rise time setting on the Bear 1000, airway pressure never exceeded zero, preventing us from measuring the defined variables.

Comparison Among Ventilators. There were significant ($p < 0.05$) and important (> 10%) differences among ventilators at each rise time setting for all variables evaluated. The trigger pressure of the Galileo was greater ($p < 0.05$) than all other ventilators, and the Siemens trigger pressure was less ($p < 0.05$) than all other ventilators except the PB 840. The inspiratory trigger time of the Galileo was greater than all other ventilators except the Dräger, while the PB 840 and Bear 1000 had the shortest ($p < 0.05$) inspiratory trigger times. The Galileo had the greatest ($p < 0.05$) trigger-pressure time product, whereas the trigger-pressure time product of the PB 840 was less ($p < 0.001$) than the Galileo and Dräger but not less than the Siemens and Bear. The Area E% of the PB 840 was greater ($p < 0.05$) than all ventilators except the Bear. The Area E% of the Galileo was less than all ventilators except the Siemens. The peak flow of the Dräger was less ($p < 0.001$) than all ventilators except the Siemens. However, the peak flow of the Bear at minimum was less ($p < 0.001$) than all ventilators. Area E of the Dräger was less ($p < 0.001$) than that of all other ventilators. The supra-plateau expiratory pressure change of the PB 840 was less ($p < 0.001$) than that of all other ventilators. The supra-plateau expiratory pressure change of the Bear was greater ($p < 0.05$) at medium and maximum rise times than any other ventilator, whereas at minimum rise time the Galileo had the greatest supra-plateau expiratory pressure change ($p < 0.05$). The expiratory time delay of the PB 840 was lower ($p < 0.001$) than that of any other ventilator. In PS mode the expiratory time delay of the Galileo was greater ($p < 0.001$) than any other ventilator. In PA/C mode the

Table 4. Inspiratory Trigger Variables and Area E During Pressure Assist/Control at 3 Rise Time Settings

	PS	Galileo	Siemens	PB 840	Bear 1000*	Dräger
P-T (cm H_2O)	minimum†	9.07 ± 0.25‡§	4.15 ± 0.25§	4.05 ± 0.38§	—	6.28 ± 0.09§
	medium†	7.93 ± 0.25§‖	3.77 ± 0.17§	3.94 ± 0.17§	4.69 ± 0.38	6.12 ± 0.09§
	maximum†	6.45 ± 0.09‡‖	1.97 ± 0.28‡‖	2.08 ± 0.09‡‖	4.65 ± 0.53	4.48 ± 0.41‡‖
D-T (s)	minimum†	0.12 ± 0.01§	0.10 ± 0.01‡§	0.09 ± 0.00§	—	0.11 ± 0.01§
	medium†	0.11 ± 0.01§	0.09 ± 0.00§‖	0.08 ± 0.01§	0.07 ± 0.01	0.12 ± 0.01§
	maximum†	0.10 ± 0.00‡‖	0.05 ± 0.01‡‖	0.06 ± 0.01‡‖	0.08 ± 0.01	0.07 ± 0.00‡‖
D-B (s)	minimum†	0.09 ± 0.01‡§	0.11 ± 0.01‡§	0.05 ± 0.00‡§	—	0.11 ± 0.01§
	medium†	0.07 ± 0.00§‖	0.07 ± 0.01§‖	0.04 ± 0.00§‖	0.08 ± 0.01§	0.11 ± 0.01§
	maximum†	0.05 ± 0.00‡‖	0.02 ± 0.01‡‖	0.02 ± 0.00‡‖	0.04 ± 0.00‡	0.03 ± 0.01‡‖
D-I (s)	minimum†	0.22 ± 0.01‡§	0.22 ± 0.01‡§	0.14 ± 0.00‡§	—	0.22 ± 0.01§
	medium†	0.18 ± 0.01§‖	0.16 ± 0.01§‖	0.12 ± 0.01§‖	0.15 ± 0.01§	0.22 ± 0.01§
	maximum†	0.15 ± 0.00‡‖	0.07 ± 0.00‡‖	0.08 ± 0.01‡‖	0.12 ± 0.01‡	0.10 ± 0.01‡‖
Area E (cm H_2O · s)	minimum†	10.97 ± 0.31	5.12 ± 0.16	5.94 ± 0.04	—	4.07 ± 0.05§
	medium†	11.36 ± 0.36	5.37 ± 0.41	6.17 ± 0.10	5.82 ± 0.21	4.00 ± 0.04§
	maximum†	11.47 ± 0.14	5.50 ± 0.21	6.03 ± 0.35	5.92 ± 0.26	4.97 ± 0.05‡‖

PS = pressure support
PB = Puritan Bennett
*No data available for BEAR 1000 at minimum setting
P-T = trigger pressure
†$p < 0.05$ among ventilators.
‡$p < 0.05$ vs medium for specific ventilator.
§$p < 0.05$ vs maximum for specific ventilator.
‖$p < 0.05$ vs minimum for specific ventilator
D-T = inspiratory trigger time.
D-B = time to return trigger pressure to baseline.
D-I = inspiratory delay time.
Area-E = area of the airway pressure curve during exhalation.

expiratory time delay of the BEAR was greater ($p < 0.05$) than that of any other ventilator.

Inspiration Termination Criteria

Figure 10 illustrates the airway-pressure-versus-time and flow-versus-time waveforms of the Galileo and PB 840 during the 3 inspiration termination criteria settings in PS mode. There were significant ($p < 0.001$) and important (> 10%) differences between all studied expiratory variables among ventilators at each inspiration termination criteria setting (Fig. 11). There were no significant differences among the inspiratory phase variables of each ventilator when the inspiration termination criteria were

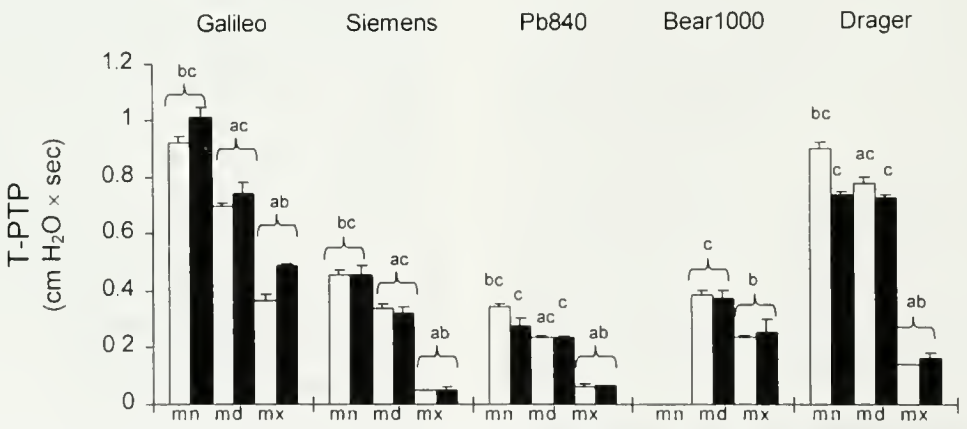

Fig. 5. Effect of minimum (mn), medium (md) and maximum (mx) inspiratory rise times on trigger-pressure time product (T-PTP) during pressure support mode (open bars) and pressure assist/control mode (closed bars) with each tested ventilator. Pb840 = Puritan Bennett 840 ventilator. a: $p < 0.05$ versus minimum. b: $p < 0.05$ versus medium. c: $p < 0.05$ versus maximum. T-PTP decreased with all ventilators in both pressure support and pressure assist/control mode as rise time increased ($p < 0.005$).

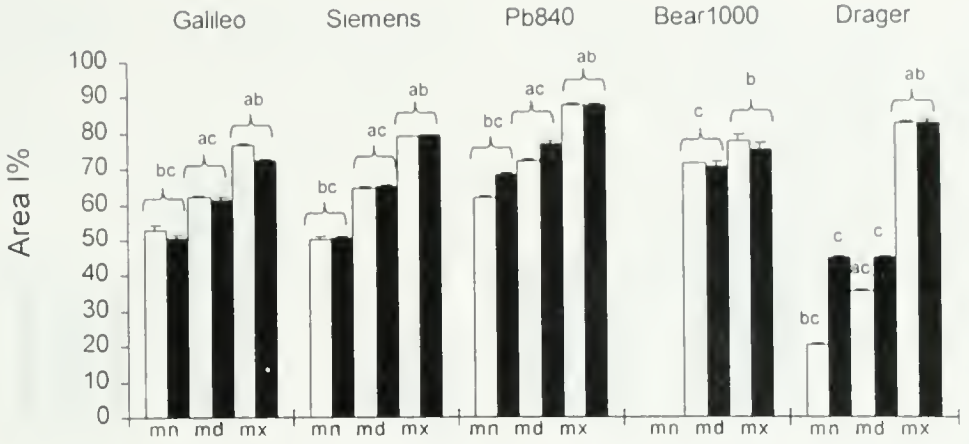

Fig. 6. Effect of minimum (mn), medium (md) and maximum (mx) inspiratory rise times on inspiratory area percent (Area I%) during pressure support mode (open bars) and pressure assist/control mode (closed bars) with each tested ventilator. Pb840 = Puritan Bennett 840 ventilator. a: $p < 0.05$ versus minimum. b: $p < 0.05$ versus medium. c: $p < 0.05$ versus maximum. Area I% increased with all ventilators in both pressure support and pressure assist/control as rise time increased ($p < 0.05$).

changed (Table 5). When the PB 840 was set at the minimum inspiration termination criteria, the ventilator prematurely cycled to exhalation (Fig. 10), and as a result Area E was negative. Consequently, supra-plateau expiratory pressure change and expiratory time delay were zero and they differed significantly ($p < 0.05$) from the medium and maximum settings (Fig. 11).

Discussion

The principle findings of this study are:

1. On all ventilators, varying the rise time produced major differences in inspiratory variables, but only small differences in expiratory variables.

2. Changing the inspiration termination criteria did not affect inspiratory variables but markedly affected all expiratory variables evaluated.

3. There were major (> 10%) differences among ventilators as inspiratory rise time and inspiration termination criteria were varied.

4. There were minimal differences between PS and PA/C on most ventilators as inspiratory rise time was altered.

Rise Time

It has been clearly established that patient effort and work of breathing are affected by the ventilator's ability to meet patient peak inspiratory demand.[12,13] If flow does not match patient demand, patient-ventilator dyssynchrony

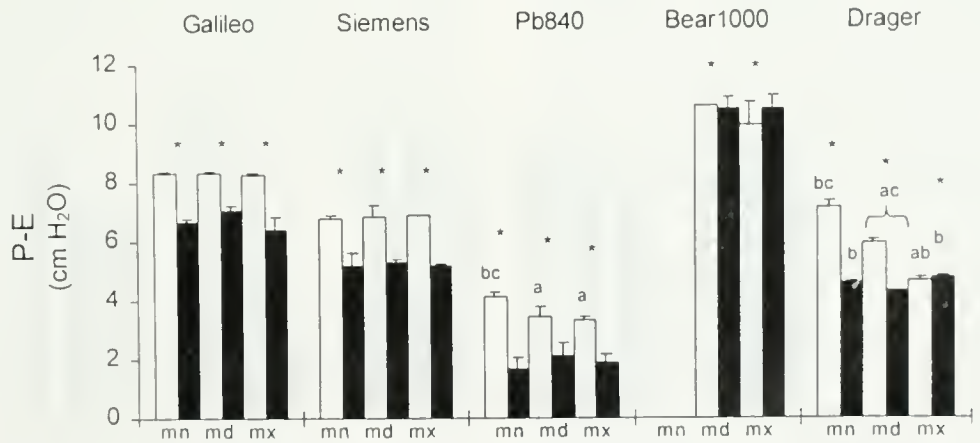

Fig. 7. Effect of minimum (mn), medium (md), and maximum (mx) inspiratory rise times on supra-plateau expiratory pressure change (P-E) during pressure support mode (open bars) and pressure assist/control mode (closed bars) with each tested ventilator. Pb840 = Puritan Bennett 840 ventilator. a: $p < 0.05$ versus minimum. b: $p < 0.05$ versus medium. c: $p < 0.05$ versus maximum. * $p < 0.05$ for pressure support versus pressure assist/control.

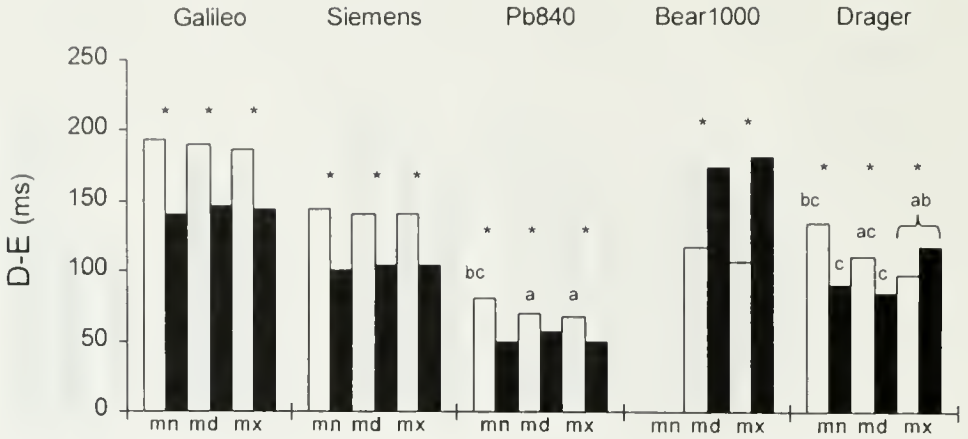

Fig. 8. Effect of minimum (mn), medium (md), and maximum (mx) inspiratory rise times on expiratory time delay (D-E) during pressure support mode (open bars) and pressure assist/control mode (closed bars) with each tested ventilator. Pb840 = Puritan Bennett 840 ventilator. a: $p < 0.05$ versus minimum. b: $p < 0.05$ versus medium. c: $p < 0.05$ versus maximum. * $p < 0.05$ for pressure support versus pressure assist/control.

occurs.[1,6] In general, PS and PA/C are better capable of meeting inspiratory demand than is volume ventilation.[1,2] Ideally, in pressure ventilation, the rise in gas flow should match the patient's demand for flow.[5] Although studies of flow synchrony during pressure ventilation suggest that many patients, because of increased ventilatory demand, require a rapid rise time, there are patients in whom a rapid rise time results in the initial system pressure exceeding the set level.[14–16] In these patients a lower rise time is preferable.

All the ventilators we tested had a large range in the levels of inspiratory variables evaluated as rise time was altered. However, the range was not linear in most ventilators. The greatest change occurred when moving from medium to maximum rise time. This was not true for the Bear, which performed poorly at minimum setting, primarily because of limited peak flow. We did not expect a change in trigger pressure as rise time was varied but did expect an increase in the time to return trigger pressure to baseline, total time delay, and trigger-pressure time product. The increase in trigger pressure as rise time decreased at a fixed lung model ventilatory drive is a result of the inability of the flow provided by the ventilator to reverse the descent in airway pressure as quickly as at a more rapid rise time. That is, activation of the flow demand system was less capable of reversing the descent in airway pressure as rise time decreased.

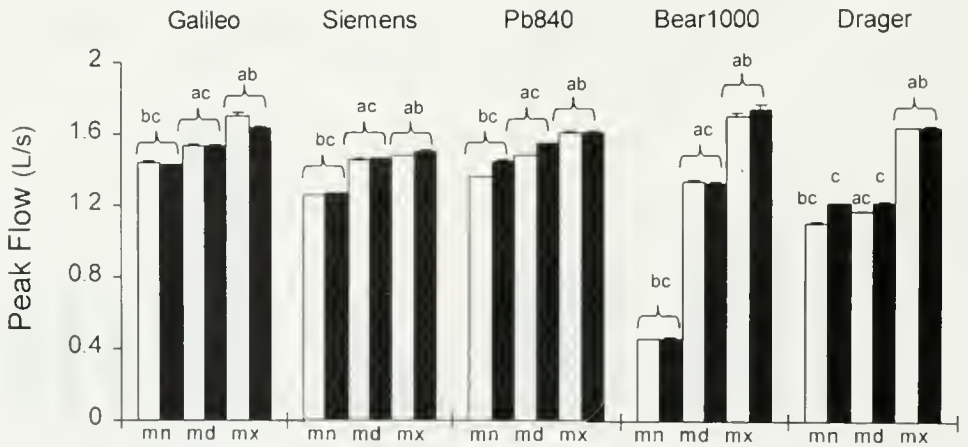

Fig. 9. Effect of minimum (mn), medium (md) and maximum (mx) inspiratory rise times on peak flow during pressure support mode (open bars) and pressure assist/control mode (closed bars) with each tested ventilator. a: $p < 0.05$ versus minimum. b: $p < 0.05$ versus medium. c: $p < 0.05$ versus maximum. Peak flow increased with all ventilators in both pressure support and pressure assist/control as rise time increased ($p < 0.05$).

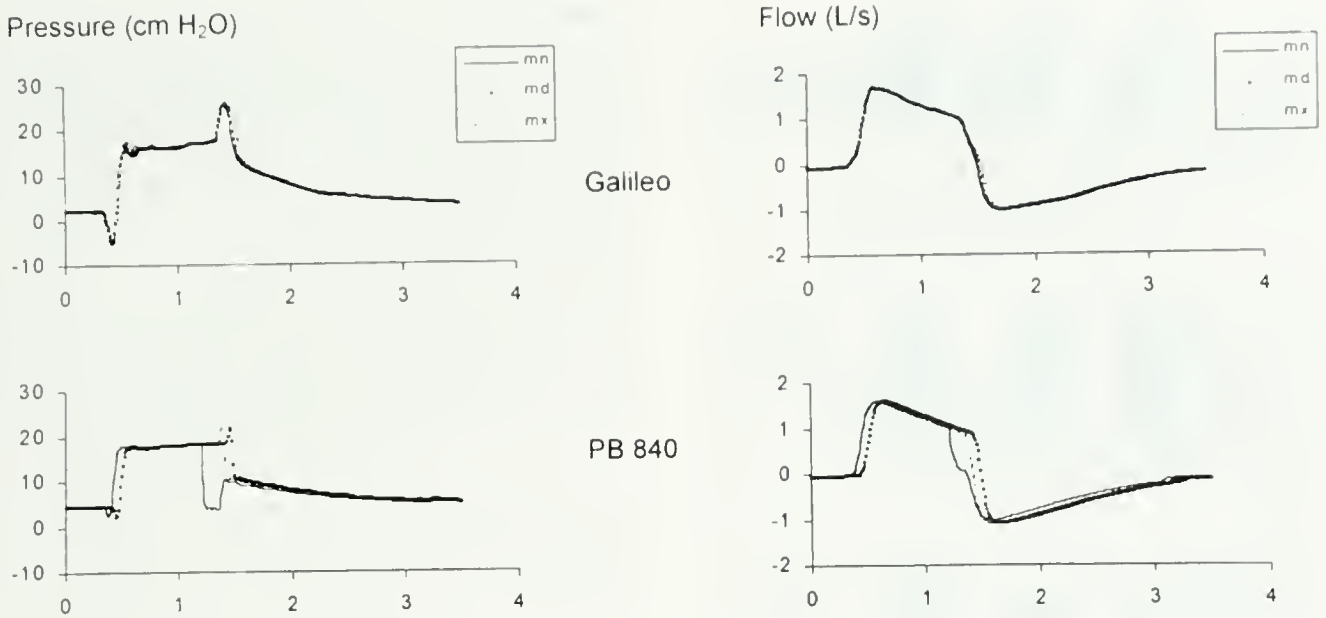

Fig. 10. Airway-pressure-versus-time (left column) and flow-versus-time (right column) waveforms of studied ventilators at minimum (mn, continuous line), medium (md, dotted line), and maximum (mx, dashed line) inspiration termination criteria. Premature exhalation occurred when minimum inspiration termination criteria were set on the Puritan Bennett 840 ventilator (PB 840).

On some of these ventilators (PB 840, Siemens 300, and Galileo), little change in peak flow was observed as rise time decreased. This is a result of 2 factors: (1) the consistent ventilatory pattern of the lung model unaffected by changes made on the ventilator (contrary to what is observed in patients), and (2) the capability of these ventilators to maintain gas delivery in spite of a change in rise time. As noted in Figures 3 and 4, airway pressure and flow waveforms changed, but peak flow changed little between minimum and maximum rise time settings.

In this evaluation, changes in rise time had minimal effect on cycling to exhalation. This is contrary to what we expected, particularly in ventilators that use a fixed percentage of peak flow as the first criterion for termination of inspiration. We anticipated, as shown by Branson et al[14] and Bonmarchard et al,[16] that as rise time was decreased, peak flow would decrease and inspiratory time would increase. However, our lung model had a fixed inspiratory time of 1 second and a fixed ventilatory drive, so expected changes in expiratory variables may not have been observable. In fact, the lung model end-inspiratory flow was high, resulting in all ventilators cycling to exhalation by pressure criteria. Our results have evaluated the ability of these ventilators to cycle when patient end-inspiratory flow exceeds the ventilator's termination criteria.

Inspiration Termination Criteria

Inspiration in PS mode is terminated by one of 3 mechanisms. The primary method is a decrease in flow. The second is a rise in pressure above the target setting. The third is inspiratory time exceeding a specific maximum duration. It has been shown by Jubran et al[9] and Parthasarathy et al[17] that cycle dyssynchrony during PS mode occurs because of activation of abdominal muscles during the inspiratory phase, increasing patient effort and the number of failed trigger efforts, whereas variability in inspiratory cycling criteria has been shown by MacIntyre and Ho[15] to have limited impact on patient-ventilator synchrony. However, Calderini et al[18] showed marked improvement in patient ventilator synchrony during noninvasive ventilation when a fixed inspiratory time during PA/C mode replaced the variable inspiratory time in PS mode.

Recently, Yamada and Du[19] mathematically modeled the factors that affect synchrony during the transition from inspiration to expiration. Specifically, they determined that the relationship of flow at the end of a patient's neural inspiratory time to peak inspiratory flow is related to 2 factors: (1) the ratio of the respiratory time constant to the patient's neural inspiratory time, and (2) the ratio of the set PS level to the maximum inspiratory muscle pressure. As a result, they reported that the ratio of patient neural inspiratory time to peak inspiratory flow may range from 1 to 85, dependent on respiratory mechanics and ventilatory drive. Thus, with set inspiration termination criteria, a patient can end inspiration before or after the ventilator reaches its termination flow. Variability in inspiration termination criteria clearly increases the probability of patient-ventilator synchrony. In our experience, problems with

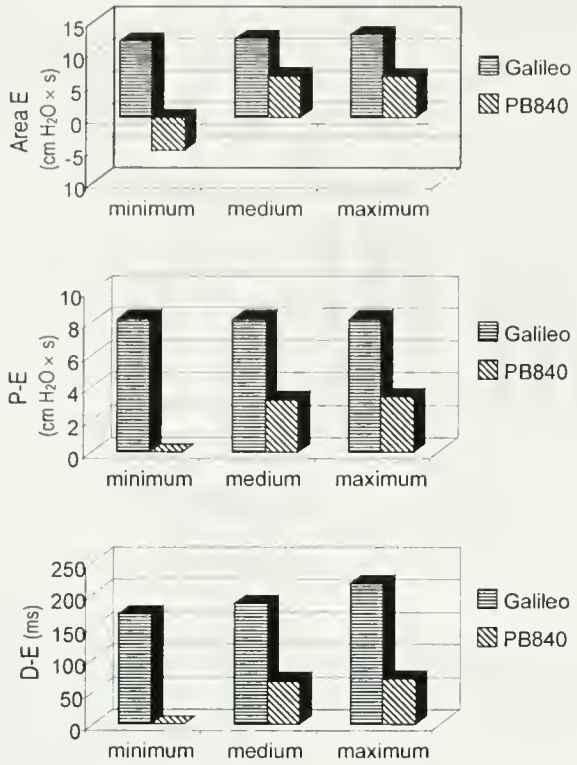

Fig. 11. Effect of inspiration termination criteria on Area E (top panel), supra-plateau expiratory pressure change (P-E) (middle panel), and expiratory time delay (D-E) (bottom panel) in pressure support mode. There were significant ($p < 0.001$) differences in Area E, P-E, and D-E between the Puritan Bennett 840 (PB840) and Galileo at every inspiration termination criteria setting.

inspiration termination synchrony are primarily observed in patients with chronic obstructive pulmonary disease ventilated with ventilators requiring very low fixed flows or very small percentages of peak flow to terminate inspiration.

As shown in Figure 11, there were marked differences between the Galileo and PB 840 in expiratory variables as inspiration termination criteria were varied. The PB 840 had a wide range of terminating flow; so much so that at the maximum setting the ventilator terminated inspiration before the lung model was ready to exhale, which is potentially a concern in patients who require careful observation of waveforms and ventilatory response to alterations in the termination criteria. On the other hand, changes to the termination criteria on the Galileo did not seem to affect the ventilator's cycling to exhalation. We have no specific explanation for that failure. No changes were made in the lung model setting between ventilators. Since the range of variability in termination criteria is similar between these 2 ventilators (see Table 2) and the peak flow delivered by these 2 ventilators differed by < 10% during this evaluation (see Table 5), we expected the Galileo and PB 840 to respond similarly.

Automation

As we have shown, all of the ventilators studied have the ability to vary rise time or both rise time and inspiration termination criteria. However, clinicians often have difficulty finding the proper settings for these variables. Proper setting of both requires careful assessment of patient synchrony with the ventilator, which is difficult to achieve without observation of the inspiratory airway pressure waveform. Ideally set rise time avoids a delay in initial gas delivery and a concavity of the initial rise in pressure (inadequate rise time) and also avoids pressure overshooting the target pressure in early inspiration (too high a rise time). Proper setting of inspiration termination criteria avoids an increase in airway pressure at end inspiration (neural inspiratory time shorter than ventilator inspiratory time). However, even if we can adjust these variables perfectly, the settings may only be perfect for the moment they are set. Any change in patient demand or neural inspiratory time will result in dyssynchrony, as in volume ventilation with a fixed inspiratory flow pattern. A

Table 5. Inspiratory Trigger Variables, Area I%, and Peak Flow During Pressure Support at 3 Inspiration Termination Criteria Settings

	ITC	Galileo	PB 840
P-T (cm H_2O)	minimum*	6.83 ± 0.25	2.19 ± 0.10
	medium*	6.83 ± 0.25	2.03 ± 0.25
	maximum*	6.61 ± 0.25	2.35 ± 0.41
D-T (s)	minimum*	0.09 ± 0.01	0.06 ± 0.01
	medium*	0.10 ± 0.01	0.06 ± 0.01
	maximum*	0.09 ± 0.01	0.06 ± 0.00
D-B (s)	minimum*	0.05 ± 0.01	0.02 ± 0.01
	medium*	0.05 ± 0.00	0.02 ± 0.01
	maximum*	0.05 ± 0.00	0.02 ± 0.00
D-I (s)	minimum*	0.14 ± 0.00	0.08 ± 0.01
	medium*	0.15 ± 0.01	0.08 ± 0.00
	maximum*	0.14 ± 0.01	0.08 ± 0.00
T-PTP (cm H_2O · s)	minimum*	0.47 ± 0.02	0.07 ± 0.00
	medium*	0.48 ± 0.03	0.07 ± 0.01
	maximum*	0.45 ± 0.02	0.08 ± 0.02
Area I%	minimum*	75.43 ± 0.45	85.37 ± 1.44
	medium*	74.86 ± 1.45	86.75 ± 0.58
	maximum*	74.94 ± 0.85	86.42 ± 0.57
Peak flow (L/min)	minimum*	1.71 ± 0.03	1.60 ± 0.01
	medium*	1.71 ± 0.03	1.60 ± 0.00
	maximum*	1.70 ± 0.02	1.60 ± 0.00

ITC = inspiration termination criteria.
PB = Puritan Bennett
P-T = trigger pressure.
D-T = inspiratory trigger time.
D-B = time to return trigger pressure to baseline
D-I = inspiratory delay time.
T-PTP = trigger pressure time product.
Area I% = inspiratory area percent.
*$p < 0.05$ among ventilators

patient's need for a specific rise time and inspiration termination criteria can vary rapidly, making it necessary to reset these variables. The ability for clinicians to adjust rise time and inspiration termination criteria is an improvement over the original design of pressure-targeted ventilation, but it may not be the final improvement. As illustrated by many investigators, there are many issues associated with dyssynchrony during pressure ventilation.[5,7,9,14–19] The next step is to automate the adjustment of rise time and inspiration termination criteria to ensure continued synchrony. We would expect the next generation of mechanical ventilators to be able to assess patient synchrony during pressure ventilation and to automatically adjust rise time and inspiration termination criteria on a breath-by-breath basis.[19]

Pressure Support Versus Pressure Assist/Control

Little difference exists between the operation of PS and PA/C as rise time is adjusted with any of the ventilators we evaluated. This is consistent with our previously published data comparing these 2 pressure-targeted modes on these same ventilators.[11]

Limitations

There are a number of limitations to this study. Most importantly, it was performed on a lung model. As a result, extrapolating the data to patients must be done cautiously, since spontaneously breathing patients have numerous inputs controlling ventilation (neural, mechanical, and chemical, as well as pain and anxiety). Second, the lung model had a fixed inspiratory time of 1.0 s. Our results may have been different if inspiratory time was different. Third, we evaluated only a single lung model peak inspiratory flow. The performance of these ventilators may differ at other peak inspiratory flows. Finally, the large differences observed in this lung model study may not reflect any variation in clinical impact with the use of these ventilators. Clinical studies are needed to identify the actual clinical impact of changes in rise time or inspiration termination criteria with any ventilator.

Conclusions

Varying the rise time settings resulted in major differences in inspiratory variables on all ventilators evaluated, but little difference in expiratory variables. The opposite occurred as inspiration termination criteria were altered. Marked changes in expiratory variables were observed, but little effect was observed on inspiratory variables. Only small (but statistically significant) differences were observed on any ventilator between PS and PA/C, but large differences were observed between ventilators during all conditions evaluated. The differences between PS and PA/C were mostly related to variables associated with expiration.

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Mean Alveolar Pressure During Constant-Flow and Constant-Pressure Inflation of Diseased Lungs

Mohamad F El-Khatib PhD and Ghassan Jamaleddine MD

INTRODUCTION: An important goal in managing mechanical ventilation is optimizing key variables such as mean alveolar pressure ($\bar{P}_A$). PURPOSE: Determine the effects of lung mechanics on $\bar{P}_A$ during constant-flow inflation (CFI) and constant-pressure inflation (CPI) in models of nonhomogenous lung disease. METHODS: We postulated a mathematical lung model consisting of 2 parallel lung units with the airways and the chest wall. Analysis was performed for step inputs of inspiratory flow and pressure while maintaining the same tidal volume, respiratory frequency, and positive end-expiratory pressure. The analysis was performed for purely restrictive, purely obstructive, and mixed lung disease. RESULTS: When simulating purely restrictive lung disease, $\bar{P}_A$ was always higher in healthy alveoli than in diseased alveoli, with constant inspiratory flow or constant inspiratory pressure. However, $\bar{P}_A$ in each alveoli was always greater during CPI than during CFI. For purely obstructive lung diseases, $\bar{P}_A$ was always lower in diseased alveoli than in healthy alveoli, with constant inspiratory flow or constant inspiratory pressure. However, $\bar{P}_A$ in each alveoli was always greater during CPI than during CFI. For mixed lung diseases with equal time constants, $\bar{P}_A$ was always higher in diseased alveoli than in healthy alveoli, with constant inspiratory flow or constant inspiratory pressure. However, $\bar{P}_A$ in each alveoli was always the same during CPI as during CFI. CONCLUSIONS: For the same tidal volume, the mean alveolar pressure in different alveoli depends on the type of disease. The difference in mean alveolar pressure between a normal and a diseased alveolus depends on the difference in time constant between those alveoli, regardless of the mode of ventilation. *Key words: mean alveolar pressure, mechanical ventilation, lung mechanics, alveoli, constant flow, constant pressure.* [Respir Care 2001;46(7):678–685]

Introduction

Constant-flow inflation (CFI) and constant-pressure inflation (CPI) are common modes of ventilatory support used in managing patients suffering respiratory failure. During CFI, breaths are delivered according to a selected flow magnitude and pattern (ie, square) and inspiration ends when a volume target is reached.[1–3] With CFI, airway pressure is the dependent variable that should be monitored and alarmed. During CPI, the ventilator increases airway pressure to an operator-defined level at the start of inspiration. Throughout the inspiratory phase, the flow decreases to maintain peak airway pressure at the set level as lung volume increases. With CPI, tidal volume (V_T) and flow are the dependent variables. Inspiratory time and maximum cycle time are also set by the clinician.[1–3]

There is surprisingly little data comparing CFI to CPI in terms of gas exchange and oxygenation[4,5] when the same V_T is used. Numerous mathematical, electrical, and mechanical models have been proposed to simulate respiratory system behaviour in response to specific settings or characteristics of a certain mode of ventilation.[6–8] This was usually done by assessing some key output variables related to airway and alveolar pressures. However, the majority of these models have assumed the respiratory system to behave as a single compartment, with constant lumped values of resistance and compliance. Earlier work by Otis et al,[9] and more recent studies by Bates et al,[10] Chelucci et al,[11] Hotchkiss et al,[12] and Chatburn et al[13] indicated that a 2-compartment model offers unique insight relevant to the distribution of gas among lung units with unequal time constants, which is a better representation of nonhomogeneously distributed lung disease.

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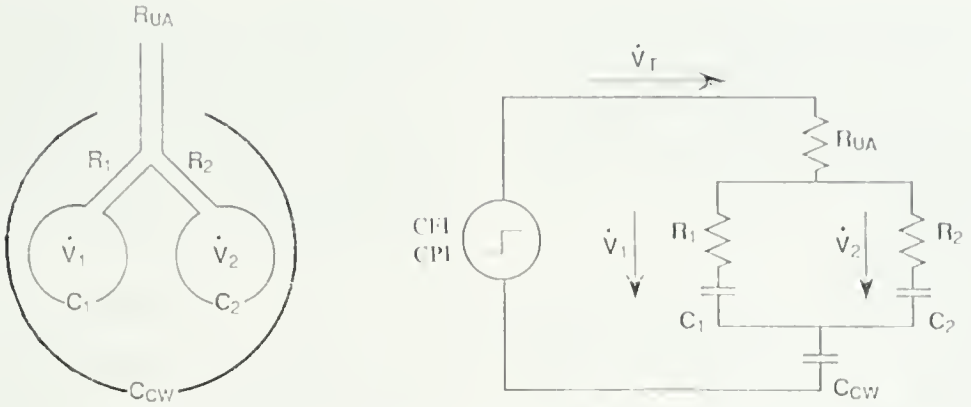

Fig. 1. Two-compartment model of the respiratory system. R_{UA} = upper airway resistance. R_1 = airway resistance of lung unit 1. R_2 = airway resistance of lung unit 2. C_1 = compliance of lung unit 1. C_2 = compliance of lung unit 2. C_{CW} = chest wall compliance. $\dot{V}_T$ = total gas flow. $\dot{V}_1$ = gas flow through lung compartment 1. $\dot{V}_2$ = gas flow through lung compartment 2. CFI = constant-flow inflation. CPI = constant-pressure inflation.

Herein we compare the mean alveolar pressure ($\bar{P}_A$), which is a determinant of arterial oxygenation to the extent that it expands the lung, at the same V_T with constant inspiratory flow and pressure applied to a mathematical model of the respiratory system. Ours is a 2-compartment model, which allows mimicking various types of lung disease, with the assumption that deflation occurs passively.

Methods and Computational Theory

Figure 1 presents a simplified analytical model of the respiratory system. It incorporates 2 parallel lung units that could represent whole lungs or neighbouring alveoli. Each lung unit has its individual compliance (C_1, C_2) connected, in series, with a resistance (R_1, R_2). The parallel lung units are connected in series with a proximal or upper-airway resistance (which might correspond to any immediately proximal common branch such as an endotracheal tube or an alveolar duct), and a chest-wall compliance. The chest wall is assumed to have no resistance; however, this assumption has no practical relevance to our model, because chest-wall resistance can be considered as part of a large resistance that includes upper-airway resistance. This model assumes that all resistances and compliances remain constant throughout the inspiratory phase. This assumption is probably valid as long as V_T and flows are normal for areas near the alveolar level, where turbulence, which could cause resistance to be nonlinear, is unlikely.

$\bar{P}_A$ can be expressed by the following equation:[7,8]

$$\bar{P}_A = \bar{P}_{aw} + \dot{V}_E(R_E - R_I) \quad (1)$$

wherein $\bar{P}_{aw}$ is mean airway pressure, $\dot{V}_E$ is minute ventilation, R_E is expiratory airway resistance, and R_I is inspiratory airway resistance. When R_I and R_E are equal, $\bar{P}_A$ will equal $\bar{P}_{aw}$. $\bar{P}_{aw}$ and $\dot{V}_E$ both depend greatly on the mode of mechanical ventilation (ie, the driving pressure or volume).

Mathematical Representation of the Model

The mathematical representation of the model was previously described in more detail.[13] It uses the previously described model equations and derivations[13] but expands to emphasize mean lung pressures rather than simply lung flows and volumes, plus the addition of unequal inspiratory and expiratory resistances. It takes into consideration:

- The equation of motion of the respiratory system,
- That two or more parallel components experience the same pressure drop, and
- That 2 or more series components share the same gas flow.

The 2-compartment model can be represented by the following equations:

$$\Delta P_{TR}(t) = \dot{V}_{tot}(t) \times f(R_1, C_1, R_2, C_2, R_{UA}, C_{CW}) \quad (2)$$

$$\dot{V}_{tot}(t) = \dot{V}_1(t) + \dot{V}_2(t) \quad (3)$$

$$\dot{V}_1(t) \times f(R_1, C_1) = \dot{V}_2(t) \times f(R_2, C_2) \quad (4)$$

wherein ΔP_{TR} is the pressure drop across the respiratory system, $\dot{V}$ is the gas flow through either lung compartment (ie, $\dot{V}_1$ and $\dot{V}_2$) or total gas flow ($\dot{V}_{tot}$), R_{UA} is upper-airway resistance, and C_{CW} is chest-wall compliance. The function f is a mathematical expression function of both airway resistance and lung compliance and represents the compartment impedance.[13] Equation 2 indicates that pressure is the product of flow and impedance, the latter rep-

resented as a generalized function of the various resistances and compliance listed. Equation 3 reflects that the total flow into the lungs is the sum of the flows into the 2 compartments. Equation 4 reflects that the pressure drop across lung unit 1 is the same as that across lung unit 2, because, as indicated in Equation 1, pressure is the product of flow and impedance.

Constant Flow Inflation

During CFI, the flow waveform was considered rectangular (ie, constant flow during inspiratory phase). With a constant inspiratory flow substituted for $\dot{V}_{tot}$ in Equations 2 and 3, the set of equations is solved for individual flows (ie, $\dot{V}_1(t)$ and $\dot{V}_2(t)$ and for $\Delta P_{TR}(t)$). Individual volumes (ie, $V_1(t)$ and $V_2(t)$) are then derived by integrating the individual flow signals over time.[13] The pressure drops across each lung unit ($\Delta P_{L1}(t)$ and $\Delta P_{L2}(t)$) and the respective mean airway pressures ($\bar{P}_{aw1}(t)$ and $\bar{P}_{aw2}(t)$) are then derived (see Appendix).

Finally, the resulting $\bar{P}_A$ are determined using Equation 1:

$$\bar{P}_{A1}(t) = \left(\int_0^{TI} \Delta P_{L1}(t)dt + PEEP \times T_I\right)/T_{tot} + V_1(t) \times RR \times (R_E - R_I) \quad (5)$$

$$P_{A2}(t) = \left(\int_0^{TI} \Delta P_{L2}(t)dt + PEEP \times T_E\right)/T_{tot} + V_2(t) \times RR \times (R_E - R_I) \quad (6)$$

wherein RR is respiratory rate, T_{tot} is total respiratory cycle time (inspiratory time [T_I] + expiratory time [T_E]), and PEEP is positive end-expiration pressure.

Constant Pressure Inflation

During CPI with a rectangular pressure waveform, the transrespiratory pressure (P_{TR} = pressure at the airway opening – pressure at the body surface) stays constant throughout the inspiratory phase. With this fact, the set of Equations 2–4 is solved for $\dot{V}_{tot}(t)$, $\dot{V}_1(t)$, and $\dot{V}_2(t)$, which are then integrated over time to derive the corresponding volumes ($V_{tot}(t)$, $V_1(t)$, and $V_2(t)$).[13] Next, $\Delta P_{L1}(t)$, $\Delta P_{L2}(t)$, $\bar{P}_{aw1}(t)$, and $\bar{P}_{aw2}(t)$ are determined (see Appendix). Finally, $P_{A1}(t)$ and $P_{A2}(t)$ are derived using Equations 5 and 6.

Disease Simulation

Airway resistances and lung compliances that simulate pure unilateral restrictive (ie, $R_1 = R_2$ and $C_1 \neq C_2$), obstructive (ie, $R_1 \neq R_2$ and $C_1 = C_2$), and mixed (ie, $R_1 < R_2$ and $C_1 > C_2$) lung diseases were analyzed (Table 1). Also, R_E was approximately twice as large as R_I.

During CFI, the step change in inspiratory flow was set at 60 L/min or 1 L/s. During CPI, the step change in inspiratory pressure was set so as to yield the same V_T as during CFI.

A respiratory rate of 12 breaths/min and PEEP of 5 cm H_2O were used for the simulations.

Results

The Case Where $R_1 = R_2$ and $C_1 \neq C_2$

When simulating a purely restrictive unilateral lung disease (ie, $C_2 < C_1$), the $\bar{P}_A$ in each alveoli was higher during pressure-controlled ventilation than during volume-controlled ventilation (ie, $\bar{P}_{A1\text{-}CPI} > \bar{P}_{A1\text{-}CFI}$ and $\bar{P}_{A2\text{-}CPI} > \bar{P}_{A2\text{-}CFI}$) (see Fig. 1). $\bar{P}_A$ was always higher in the healthy alveoli (ie, $\bar{P}_{A2} < \bar{P}_{A1}$), regardless of whether CPI or CFI was used (see Fig. 1).

The Case Where $R_1 \neq R_2$ and $C_1 = C_2$

When simulating a purely obstructive lung disease (ie, $R_2 > R_1$), $\bar{P}_A$ in each alveolus was higher during pressure-controlled ventilation than during volume-controlled ventilation (ie, $\bar{P}_{A1\text{-}CPI} > \bar{P}_{A1\text{-}CFI}$ and $\bar{P}_{A2\text{-}CPI} > \bar{P}_{A2\text{-}CFI}$) (Fig. 2). $\bar{P}_A$ was always lower in the healthy alveoli (ie, $\bar{P}_{A1} < \bar{P}_{A2}$), regardless of whether CPI or CFI was used (see Fig. 2).

The Case Where $R_1 < R_2$ and $C_1 > C_2$ but the Time Constants Are Equal

When the airway resistance and lung compliance of one compartment lung unit were varied so as to keep the time constants of each compartment the same, $\bar{P}_A$ was always lower in the healthy alveoli, regardless of whether CFI or CPI was used (Fig. 3). Moreover, $\bar{P}_A$ in each alveoli was

Table 1. Values for Respiratory System Mechanics Used in the Graphical Illustration of the 3 Models of Lung Disease

Model	R_1	C_1	R_2	C_2	R_{UA}	C_{CW}
Restrictive	2	0.1	2	0.05	2	0.2
Obstructive	2	0.1	8	0.1	2	0.2
Equal τ	2	0.1	8	0.025	2	0.2

Resistances are in cm $H_2O \cdot s/L$
Compliances are in L/cm H_2O.
R_1 = resistance of lung unit 1
C_1 = compliance of lung unit 1
R_2 = resistance of lung unit 2
C_2 = compliance of lung unit 2
R_{UA} = resistance of the upper airway
C_{CW} = resistance of the chest wall
τ = time constant

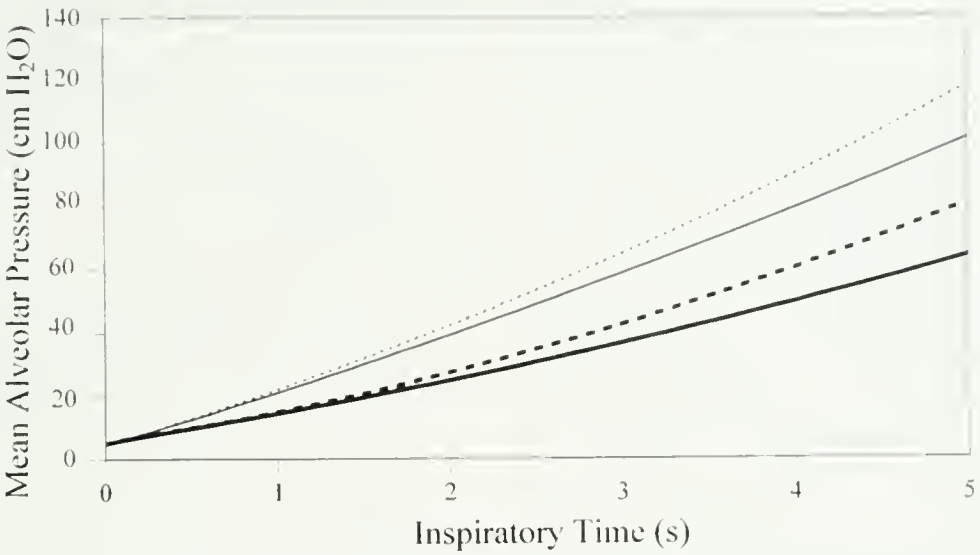

Fig. 2. Mean alveolar pressures for a 2-compartment model with equal airway resistances but different compliances (as in restrictive lung disease). Values for model variables are presented in Table 1. Thin solid line = healthy lung unit with constant-flow inflation. Thick solid line = diseased lung unit with constant-flow inflation. Thin dashed line = healthy lung unit with constant-pressure inflation. Thick dashed line = diseased lung unit with constant-pressure inflation.

the same during pressure-controlled ventilation as during volume-controlled ventilation (ie, $\bar{P}_{A1\text{-}CPI} = \bar{P}_{A1\text{-}CFI}$ and $\bar{P}_{A2\text{-}CPI} = \bar{P}_{A2\text{-}CFI}$) (Fig. 4).

Discussion

In this study we analyzed a 2-compartment model of the respiratory system for $\bar{P}_A$ under constant inspiratory pressure and flow. Our analysis suggested that for the same V_T, constant inspiratory pressure inflation would result in a higher $\bar{P}_A$ than constant inspiratory flow inflation for both obstructive and restrictive lung disease. Furthermore, our findings suggest that the distribution of $\bar{P}_A$ within lung units would primarily be influenced by the time constants of the lung units.

There are important features of constant inspiratory flow and pressure breaths that can explain the differences observed in $\bar{P}_A$. Rapid initial increase in gas flow of a constant inspiratory pressure breath will result in a faster rise time of airway pressure; thus maximum airway pressure can be reached earlier during constant inspiratory pressure, whereas maximum airway pressure is achieved at end of inspiration with constant inspiratory flow.[1,5,13,14] During constant inspiratory flow, the resistance component remains essentially constant throughout inspiration, including the period at end inspiration when the compliance component is greatest. In constant pressure inflation, since flow decreases during inspiration, the resistance component of airway pressure decreases as the compliance component increases, resulting in an overall stabilization of peak airway pressure.

Our results indicate that time constants of different lung units play a major role in establishing $\bar{P}_A$ inside the lungs. Previous reports[15,16] have shown that the maximum regional pressures among lung units with heterogeneous ventilatory time constants are exaggerated and unlimited, and greatly dependent on the distribution of $\dot{V}_I$, and, thus, the mode of mechanical ventilation. Those findings are in accordance with our findings. We showed that whenever the time constants of 2 neighbouring lung units are the same, the distribution of $\bar{P}_A$ was identical, regardless of the mode of ventilation applied.

Although our study was not intended to look at the effect of inspiratory time, our results reflect a more dramatic difference in the distribution of $\bar{P}_A$ whenever inspiratory time was greater than half the total respiratory cycle time (ie, inspiration-expiration ratio $> 1:1$). This is the range where the development of intrinsic PEEP is most likely. Thus, our findings confirm those reported by Hotchkiss et al[12] regarding the effect of intrinsic PEEP on the inspiratory distribution of ventilation and ultimately on gas exchange.

There is increasing interest in the use of $\bar{P}_{aw}$ and $\bar{P}_A$ to guide alterations in ventilator settings,[17-19] since changes in $\bar{P}_{aw}$ and $\bar{P}_A$ play an important role in mediating ventilator-induced alterations in gas exchange, cardiovascular function, and barotrauma/volutrauma. The importance of $\bar{P}_{aw}$ as a determinant of oxygenation was first emphasised

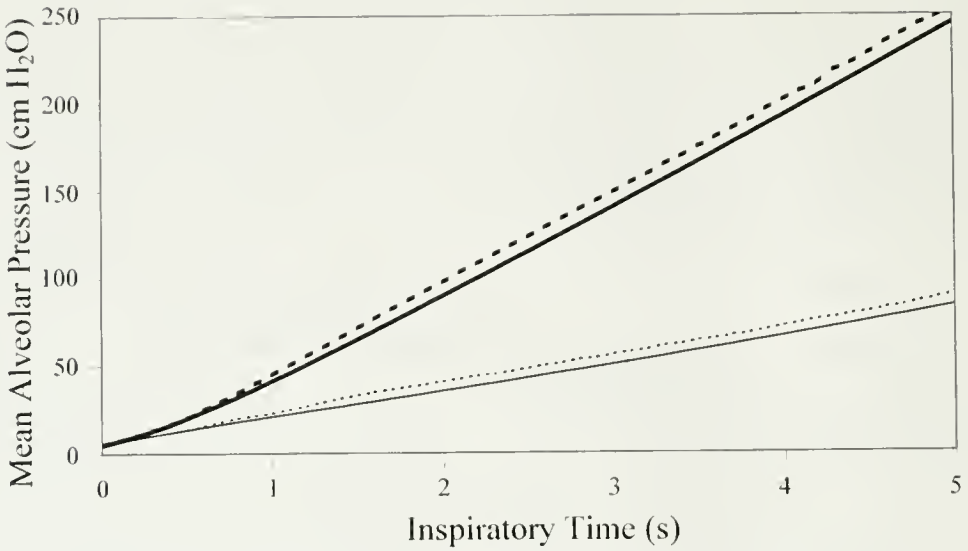

Fig. 3. Mean alveolar pressures for a 2-compartment model with equal compliances but different airway resistances (as in obstructive lung disease). Values for model variables are presented in Table 1. Thin solid line = healthy lung unit with constant-flow inflation. Thick solid line = diseased lung unit with constant-flow inflation. Thin dashed line = healthy lung unit with constant-pressure inflation. Thick dashed line = diseased lung unit with constant-pressure inflation.

by Boros et al, who studied ventilator-supported neonates and premature lambs with severe lung diseases.[20,21] They found that improvements in oxygenation were directly related to increases in $\bar{P}_{aw}$, which was thought to be due to recruitment of collapsed alveoli and/or the redistribution of lung fluid.

Despite the fact that increasing $\bar{P}_{aw}$ and $\bar{P}_A$ can improve oxygenation, there are recent reports showing that those increases can also cause lung injury and delay the recovery of injured lung tissue by stretching the alveolar walls and increasing vascular resistance and vascular transmural pressure.[22] We observed in our findings that both modes of inspiratory inflation resulted in increasing alveolar pressure in the normal lung units when simulating a purely restrictive lung disease. This uneven distribution of pressure could explain the perpetuation of damage induced by

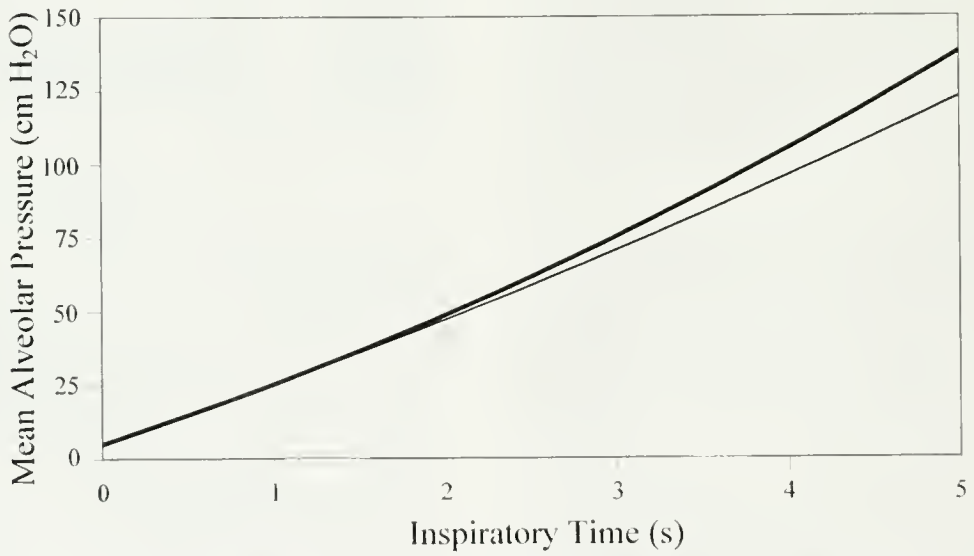

Fig. 4. Mean alveolar pressures for a 2-compartment model with equal time constants. Values for model variables are presented in Table 1. Thin line = healthy lung unit with constant-flow inflation and constant-pressure inflation. Thick line = diseased lung unit with constant-flow inflation and constant-pressure inflation.

mechanical ventilation. The normal lung is more predisposed to the over-stretch mechanical insult. Furthermore, the heterogeneous appearance of hyperinflated and atelectatic parenchyma observed on chest computed tomography in acute lung injury after several days of mechanical ventilation could be attributed to this uneven distribution of $\bar{P}_A$. In contrast, when simulating a purely obstructive lung disease where only regional airway resistances were changed, both modes of inspiratory inflation resulted in increasing alveolar pressure in the diseased lung units.

During positive pressure ventilation, $\bar{P}_{aw}$ is the pressure measured at the airway opening, averaged over the entire respiratory cycle, and reflects the average pressure applied by the ventilator.[1,3] Under these same conditions, $\bar{P}_A$ is the average pressure acting to distend the alveoli against the combined recoil of lung and chest wall.[14] Thus, $\bar{P}_A$ corresponds directly to alveolar recruitment, shunt reduction, and blood oxygenation.[14] Since $\bar{P}_A$ cannot be measured directly during the tidal cycle, it can be estimated from a mathematical relationship of P_A to P_{aw}. $\bar{P}_A$ exceeds $\bar{P}_{aw}$ by an amount that depends both on $\dot{V}_I$ and the difference between R_I and R_E. Furthermore, $\dot{V}_E$ and distribution of V_T depends greatly on the mode of mechanical ventilation and the health or disease status of the lungs.[9,13] The current analysis presented an opportunity to directly assess $\bar{P}_A$ rather than using $\bar{P}_{aw}$ as a surrogate of $\bar{P}_A$. This was achieved by taking into consideration the distribution of the $\dot{V}_I$ and the status of the lungs by simulating different types of obstructive and restrictive lung diseases.

In contrast to other studies, which used 1-compartment models,[23–25] this report and another report[12] analyzed a 2-compartment model of the respiratory system. A 2-compartment model should be superior to a 1-compartment model in accurately describing and mimicking health and pathological conditions of the lungs. By representing the airways and parenchyma of each lung unit separately, as well as the upper airway resistance and the chest wall compliance, the current model can simulate any disease entity that can affect either 1 part of the model or any other combination of 2 or more parts. However, we must bear in mind the limitations of any model study. Even a 2-compartment model is a gross oversimplification of the respiratory system. It does not take into account the compliance of the airways and inertness, the dependence between lung regions mediated through lung parenchyma and chest wall, or any nonlinear behaviour of resistance or compliance.

Although chest-wall resistance is not included in this model, this omission has no particular relevance, since chest-wall resistance can be considered as a common resistance together with airway resistance. As the main purpose of this study was to compare the behavior of $\bar{P}_A$ during constant inspiratory pressure and flow, only the essential parameters were varied, such as the 2 compartments' mechanics (R_1, R_2, C_1, and C_2), to simulate different types of disease. The expiratory resistance was not varied; however, it was set to be twice the inspiratory resistance. Although relevant and informative, a full range of respiratory rates, inspiratory and expiratory times, and V_T were not explored. The main purpose of the current study was to assess the effect of lung disease (ie, nonhomogenous changes in lung compliance and/or airway resistance) on the distribution of $\bar{P}_A$ during CFI and CPI.

Our analysis is limited in several ways, and its applicability to actual mechanical ventilation is uncertain because the relevant properties of real respiratory systems that could be used at the bedside to guide the choice of ventilation mode are not, at present, determinable, and the distributions of ventilation and perfusion, not just ventilation, are the important determinants of gas exchange and oxygenation. Nevertheless, our analysis should provide a basis for generalizations about the potential for barotrauma/volutrauma and a theoretical foundation for clinical studies.

As the main purpose of this study is to develop a mathematical model that can describe the behavior of $\bar{P}_A$ in nonhomogenous lung disease, a mechanical test lung analogue was not used. However, this does not negate the need for validating the current findings in a mechanical test lung model, which could be the essence of future investigation.

In conclusion, for the same V_T, CPI can result in higher $\bar{P}_A$ than CFI and therefore might result in better oxygenation than CFI in nonhomogeneous lung diseases. However, the higher $\bar{P}_A$ with CPI in the normal lung units with unilateral and nonhomogeneous restrictive lung disease might increase the risk of lung injury and cardiovascular deterioration by mechanical ventilation. Theoretically, the time constants of different lung units could be the most crucial factor differentiating lung ventilation and oxygenation. An increase in airway resistance could be compensated by a decrease in lung compliance, keeping the time constants the same and thus keeping the $\bar{P}_A$ difference between a normal and a diseased lung unit the same.

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APPENDIX

Previously Derived Variables

(See Reference 13 for actual equations)

$f(R_1, C_1)$	This is a mathematical expression function of the resistance and compliance of lung unit 1. It represents the compartment impedance.
$f(R_2, C_2)$	This is a mathematical expression function of the resistance and compliance of lung unit 2. It represents the compartment impedance.
$f(R_1, R_2, C_1, C_2, R_{UA}, C_{CW})$	This is a mathematical expression function of the resistances and compliances of the total respiratory system, including upper airway and chest wall. It represents the total respiratory system impedance.
$\dot{V}(t)$	Gas flow in a lung compartment.
$V(t)$	Gas volume delivered to a lung compartment. The volume is related to the gas flow by the time factor (ie, the volume is the result of a certain flow maintained for a certain time). In our study, the volume is derived by integrating the gas flow over inspiratory time.
$\Delta P_L(t)$	Pressure drop across a lung compartment. It is a function of both the gas flow through the lung compartment and the compartment's impedance.

Newly Derived Variables

$\overline{P}_{aw}(t)$	Mean airway pressure. This is a function of many variables, such as peak airway pressure, positive end-expiratory pressure, and inspiratory and expiratory time. It is the average level of pressure seen over the total respiratory cycle and reflected by the average area under the pressure curve. As such it is derived by integrating the pressure drop across the lung compartment and dividing it by the total respiratory cycle time (T_{tot}, which is the sum of inspiratory and expiratory times).
$\overline{P}_{Al}(t)$	Mean alveolar pressure. Represented by Equation 1 in the text.

Static and Dynamic Pressure-Volume Curves Reflect Different Aspects of Respiratory System Mechanics in Experimental Acute Respiratory Distress Syndrome

Alexander B Adams MPH RRT FAARC, Nahit Çakar MD, and John J Marini MD

INTRODUCTION: A lower inflection point, an upper inflection (or deflection) point, and respiratory system compliance can be estimated from an inspiratory static pressure-volume (SPV) curve of the respiratory system. Such data are often used to guide selection of positive end-expiratory pressure (PEEP)/tidal volume combinations. Dynamic pressure-volume (DPV) curves obtained during tidal ventilation are effortlessly displayed on modern mechanical ventilator monitors and bear a theoretical but unproven relationship to the more labor-intensive SPV curves. OBJECTIVE: Attempting to relate the SPV and DPV curves, we assessed both curves under a range of conditions in a canine oleic acid lung injury model. METHODS: Five mongrel dogs were anesthetized, paralyzed, and monitored to assure a stable preparation. Acute lung injury was induced by infusing oleic acid. SPV curves were constructed by the super-syringe method. DPV curves were constructed for a range of PEEP and inspiratory constant flow settings while ventilating at a frequency of 15 breaths/min and tidal volume of 350 mL. Functional residual capacity at PEEP = 0 cm H_2O was measured by helium dilution. The change in lung volume by PEEP at 8, 16, and 24 cm H_2O was measured by respiratory inductance plethysmography. RESULTS: The slope of the second portion of the DPV curve did not parallel the corresponding slope of the SPV curve. The mean lower inflection point of the SPV curve was 13.2 cm H_2O, whereas the lower inflection point of the DPV curve was related to the prevailing flow and PEEP settings. The absolute lung volume during the DPV recordings exceeded ($p < 0.05$) that anticipated from the SPV curves by (values are mean ± SEM) 267 ± 86 mL, 425 ± 129 mL, and 494 ± 129 mL at end expiration for PEEP = 8, 16, and 24 cm H_2O, respectively. CONCLUSIONS: The contours of the SPV curve are not reflected by those of the DPV curve in this model of acute lung injury. Therefore, this study indicates that DPV curve should not be used to guide the selection of PEEP/tidal volume combinations. Furthermore, an increase in end-expiratory lung volume occurs during tidal ventilation that is not reflected by the classical SPV curve, suggesting a stable component of lung volume recruitment attributable to tidal ventilation, independent of PEEP. *Key words: pressure-volume curves, pulmonary mechanics, mechanical ventilation, ventilatory strategy, oleic acid-induced lung injury, acute respiratory distress syndrome, ARDS, monitoring.* [Respir Care 2001;46(7):686–693]

Introduction

Static inspiratory pressure-volume (SPV) curves have been used in research and clinical environments to select a best combination for positive end-expiratory pressure (PEEP) and tidal volume (V_T) to use in ventilating patients with acute lung injury. Three features of these curves are thought to be helpful: the lower inflection point (LIP), the

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upper inflection point (UIP), and the slope of the linear segment (a measure of respiratory system compliance).[1] Whereas the airway pressure-volume plot can be displayed effortlessly by modern intensive care unit ventilators, obtaining SPV curves disrupts care delivery, imposes a hazard of hypoxemia, and requires considerable time to complete.

When constant flow is provided during inspiration, resistive pressure is relatively constant, theoretically offsetting the alveolar pressure-volume relationship by a fixed amount while preserving the shaping information. At conventional rates of flow, however, multiple potential problems are encountered: pressures are required not only to overcome resistance and viscoelastance forces, but also to deliver flow to a lung with airway secretions and multiple compartments with disparate time constants. We therefore question the validity of the shaping information of the dynamic pressure-volume (DPV) curves for selecting PEEP and V_T combinations.

Two recent studies reported the use of a slow flow method (3, 9, and 15 L/min) of generating pressure-volume curves.[2,3] This quasi-dynamic method produced pressure-volume curves that were comparable to conventional super-syringe method curves. In the 1980s, Suratt et al reported a "pulsed" method for measuring dynamic compliance that measured the slope of the inspiratory airway pressure curve during constant flow ventilation.[4,5] The compliance measured by this method compared favorably to the usual calculation of compliance (the change in lung volume [ΔV] divided by plateau pressure minus total PEEP). Therefore, these studies could be used to justify the use of dynamic information (DPV curves?) to measure static (SPV) conditions. More recently, Ranieri et al found changes in curve contour and difficulties in identifying LIP and UIP in DPV curves under truly dynamic conditions.[6] Therefore, to further test the differences between SPV and DPV, we compared SPV and conventional flow DPV (using flows typical for tidal ventilation) by constructing and comparing SPV and DPV under a range of conditions in a canine model of acute lung injury.

Methods

Five adult mixed-breed dogs of either sex (mean weight 25.1 ± 3.4 kg) were included in this study, using a protocol approved by the Institutional Animal Care and Use Committee of Regions Hospital. The dogs were paralyzed and deeply sedated throughout the experimental protocol. Mechanical ventilation was initiated in constant-flow, volume-cycled mode (ventilator model 7200, Mallinckrodt, Carlsbad, California). To assure the safety and stability of the preparation, arterial pH, arterial partial pressure of carbon dioxide (P_{aCO_2}), arterial partial pressure of oxygen (P_{aO_2}), blood pressure, central venous pressure, and pulmonary artery wedge pressure were measured throughout the study period. Airway opening pressure and air flow were measured between the circuit Y-piece and the endotracheal tube by a lateral pressure tap and pneumotachometer (Hans Rudolph Inc, Kansas City, Missouri). The pressure tap and pneumotachometer were connected to differential pressure transducers (model MP-45, Validyne, Northridge, California). The signals were then demodulated, amplified, and graphed continuously on a chart recorder (model 9500, Astromed, West Warwick, Rhode Island). Lung volume level was measured continuously with a respiratory inductive plethysmograph (Respigraph, NIMS, Miami Beach, Florida). Airway pressure and flow signals were transferred to a Macintosh-compatible computer and digitized at 50 Hz using commercially available software (LabView, National Instruments, Austin, Texas). The flow signal was integrated to produce a continuous signal of changing lung volume.

Lung injury was induced by infusing oleic acid (0.09 mL/kg) into the pulmonary artery over a 15-minute period, with lateral positioning of the animal to create an even distribution of injury. Ventilation was continued for 4 hours before commencing data collection. We then obtained SPV curves and DPV curves as described in the following section. Functional residual capacity (FRC) was measured by helium dilution (FRC(He)). A valved 1.0 L syringe was filled with a measured concentration of helium (helium analyzer, Collins Inc, Braintree, Massachusetts). After a disconnection from the ventilation for 20 seconds (zero end-expiratory pressure, ZEEP), the syringe was connected to the endotracheal tube and 10 volumes of approximately 500 mL were injected and withdrawn. The final diluted concentration of helium was measured, and FRC was calculated by a standard dilution calculation—subtracting connector-valve dead space. FRC(He) was determined in triplicate and the results were averaged to calculate a mean FRC. Lung volume above FRC was measured by respiratory inductive plethysmography during a ventilator disconnection to ZEEP followed by 20 seconds of apnea.

Static Pressure-Volume Curves

Prior to the SPV measurement, the endotracheal cuff volume was filled to assure there was no leak during the procedure. Following a 20 second disconnection from the ventilator, a 3.0 L super-syringe filled with pure oxygen was connected to the endotracheal tube. The lungs were then incrementally inflated, by 50 mL steps (5–10 s between steps), until airway pressure attained 40 cm H_2O. A deflation maneuver immediately followed, using similar 50 mL deflation steps. Airway pressure and lung volume were measured during the procedure. The associated pressure and volume values were plotted to construct the SPV curves (using Deltagraph, SPSS, Chicago).

Table 1 Arterial Blood Gas and Respiratory System Mechanics Measurements Before and After Oleic Acid Injury

	pH	P_{aCO_2} (mm Hg)	P_{aO_2} (mm Hg)	$P_{\bar{v}O_2}$ (mm Hg)	$\dot{Q}_S/\dot{Q}_T$	Peak P_{aw} (cm H_2O)	Plat P_{aw} (cm H_2O)	Mean P_{aw} (cm H_2O)
Baseline	7.29 ± 0.08*	47 ± 8*	280 ± 32*	56 ± 6	0.07 ± 0.02*	11.3 ± 1.1*	9.7 ± 0.7*	4.7 ± 0.6*
OAI	7.17 ± 0.07	59 ± 15	74 ± 19	45 ± 11	0.38 ± 0.10	16.1 ± 4.1	13.5 ± 3.0	6.6 ± 1.0

Values are mean ± SD
P_{aCO_2} = arterial partial pressure of carbon dioxide.
P_{aO_2} = arterial partial pressure of oxygen
$P_{\bar{v}O_2}$ = average venous partial pressure of oxygen
$\dot{Q}_S$ = shunting flow
$\dot{Q}_T$ = total flow
Peak P_{aw} = peak airway pressure
Plat P_{aw} = plateau airway pressure
Mean P_{aw} = mean airway pressure
OAI = measured after oleic acid infusion
*$p < 0.05$ between baseline and after OAI, paired comparisons

Dynamic Pressure-Volume Curves

DPV curves were obtained while ventilating with volume-controlled ventilation at a V_T of 350 mL (12–15 mL/kg) and a frequency of 15 breaths/min to simulate dynamic tidal ventilation conditions. Using this V_T and frequency combination, no intrinsic PEEP was generated at any flow setting. Tracings were recorded at constant inspiratory flows of 10, 30, and 50 L/min and PEEP of 0, 8, 16, and 24 cm H_2O for each flow setting. A minimum of 10 consecutive tidal breaths were delivered in each condition before the stereotypical pattern for that setting was recorded. A lung volume history of 20 seconds of apnea was interposed between each change in PEEP setting. At each PEEP setting the permutations of flow settings (10, 30, and 50 L/min) were randomly selected for each DPV recording. The DPV curves were then constructed by plotting the digitized airway pressure and volume signals (from flow integration) using a graphing software program (Deltagraph, SPSS, Chicago).

Data Analysis

A graph of the overlaid SPV and DPV curves was constructed from the independently measured curves. The relative curve position was determined by the FRC and volume-above-FRC measurements. All curves were positioned above the FRC(He) level. Total FRC at each specific PEEP level was determined by adding the FRC(He) and the ΔV to ZEEP measured by respiratory inductive plethysmography (Fig. 1).

Differences between the SPV and DPV curves were examined further by comparing compliance and LIP measurements. Respiratory system compliance of the SPV curves was calculated by measuring ΔV and ΔP between the LIP and UIP of the inspiratory curves. Compliance was then calculated by $\Delta V/\Delta P$. The precise pressure change (ΔP) between 50 mL and 350 mL of the delivered V_T determined compliance (= 300 mL/ΔP). LIP for the SPV and DPV were determined by aligning a cursor on the video-display of the digitized curves at the median point of the lower inflection curve and then read as the LIP. Significance testing was by paired and unpaired *t* tests (StatView, Abacus, Berkeley, California).

Results

Effect of Oleic Acid Infusion

After oleic acid infusion, the peak, mean, and end-inspiratory plateau airway pressures increased (Table 1). Gas exchange deteriorated, as evidenced by a decrease ($p < 0.001$) in P_{aO_2}, a decrease ($p < 0.01$) in pH, an increase ($p < 0.001$) in P_{aCO_2}, and an increase ($p < 0.001$) in the ratio of shunting flow to total flow ($\dot{Q}_S/\dot{Q}_T$) (see Table 1). After the injury, the mean LIP of the SPV curve was 13.2 ± 1.6 cm H_2O.

Static Pressure-Volume Curves and Dynamic Pressure-Volume Curves

Figures 2 through 6 show SPV and DPV overlay curves for the 5 studies. In each study, increasing the flow setting shifted the inflection portion of the DPV curves to the right by an amount proportionate to flow. Though the upward slope of the DPV curve (the lung recoil pressure component?) was expected to parallel the inspiratory SPV slope, the actual DPV slope was steeper than the respective SPV slope. The DPV curves for 2 animals (Figs. 2 and 5) were highly irregular, displaying a "whip," and associated with visual evidence of secretions in the central airways at end expiration. PEEP displaced the initiation of the DPV vertically along the volume axis with respect to the SPV curve (inside the envelope of the SPV curve).

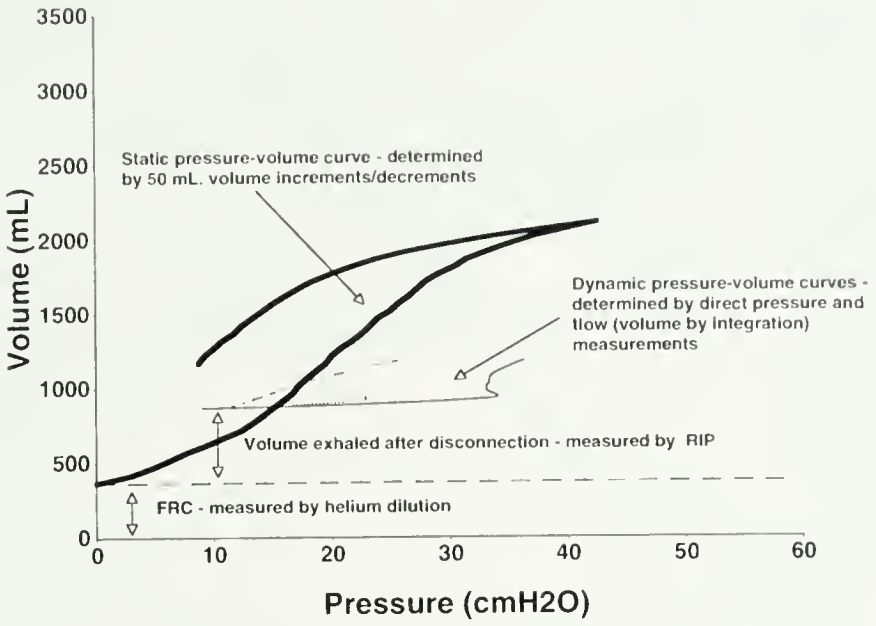

Fig. 1. Key to reading the overlay (positioning) of the static pressure-volume and dynamic pressure-volume curves. The functional residual capacity measured by helium dilution (FRC(He)) is the volume at zero end-expiratory pressure for starting both the static pressure-volume curve and the dynamic pressure-volume curve at positive end-expiratory pressure (PEEP) = 0 cm H_2O. The dynamic pressure-volume curves at other PEEP settings start at their respective PEEP levels (x position) and at FRC(He) + the change in volume (ΔV) after ventilator disconnection (y position). RIP = respiratory inductive plethysmography.

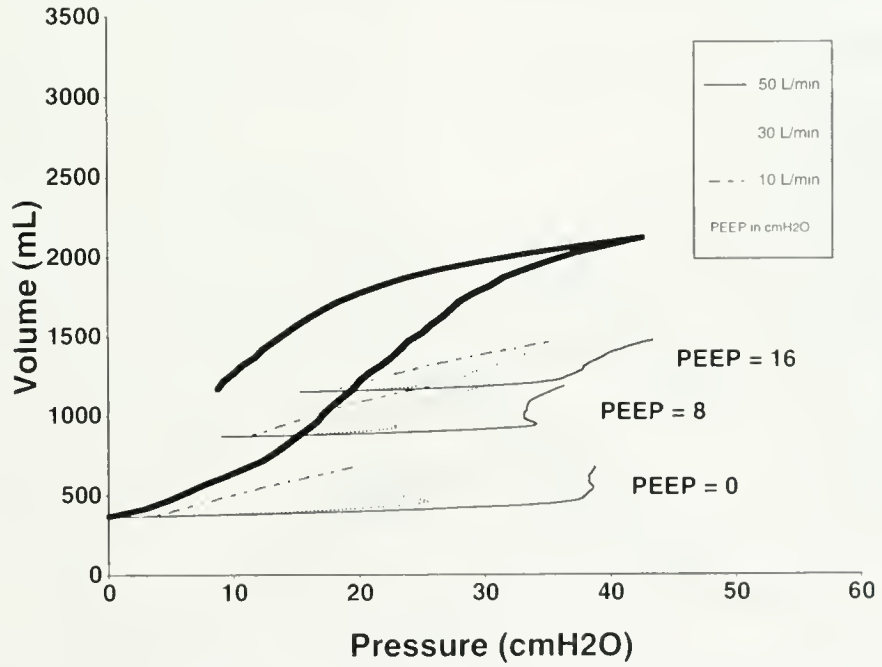

Fig. 2. Static and dynamic pressure-volume relationships for Subject 1. For this and all subsequent, the effects of positive end-expiratory pressure (PEEP) and flow variation are depicted at a constant tidal volume and respiratory frequency. Absolute lung volume is plotted as a function of dynamic (fine lines) and static (heavy line) airway pressures for an individual animal. The second (rising) portion of each dynamic inspiration is shifted in proportion to flow. In this animal note that the slopes of the dynamic pressure-volume curves are influenced by secretions that were visualized in the central airways at end expiration.

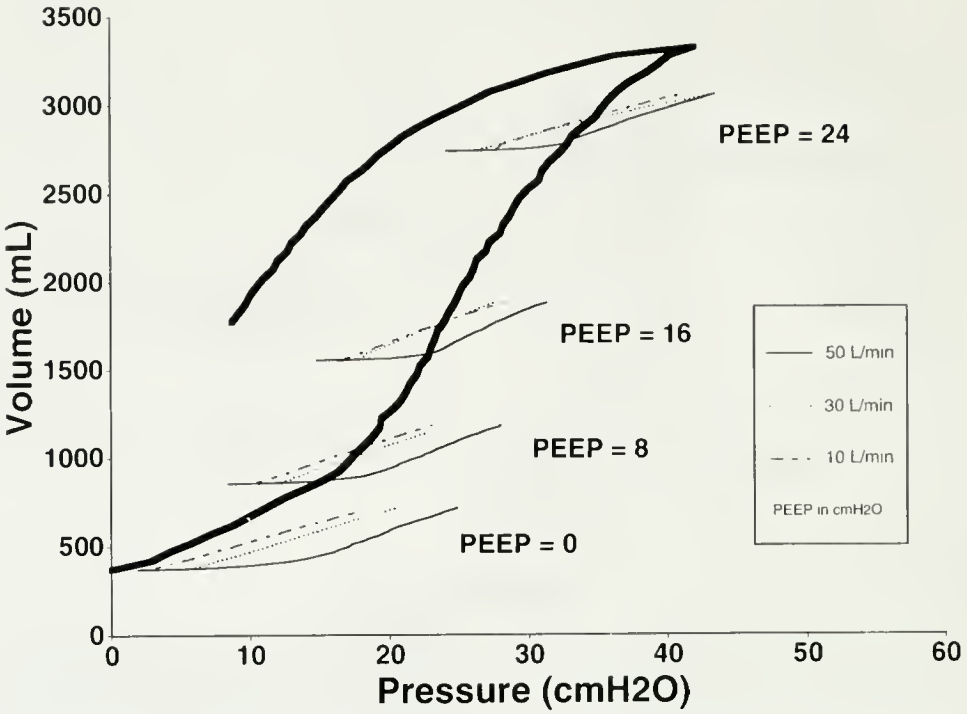

Fig. 3. Subject 2: End-expiratory (starting) points of each dynamic pressure-volume curve are displaced progressively toward the expiratory curve with rising positive end-expiratory pressure (PEEP). The inspiratory slopes of the dynamic pressure-volume curves were similar, independent of the flow setting, but differed radically from the static pressure-volume curve slope.

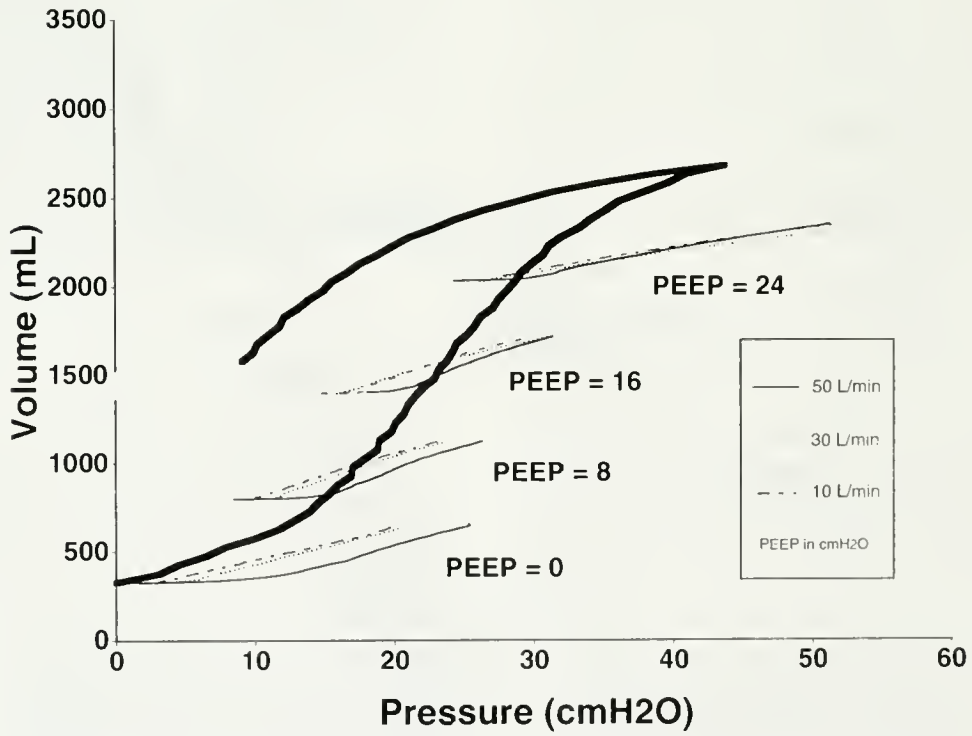

Fig. 4. Subject 3: Note the uniformity of the dynamic pressure-volume curves, despite flow variation and a disparity with the inspiratory limb of the static pressure-volume curve. PEEP = positive end-expiratory pressure.

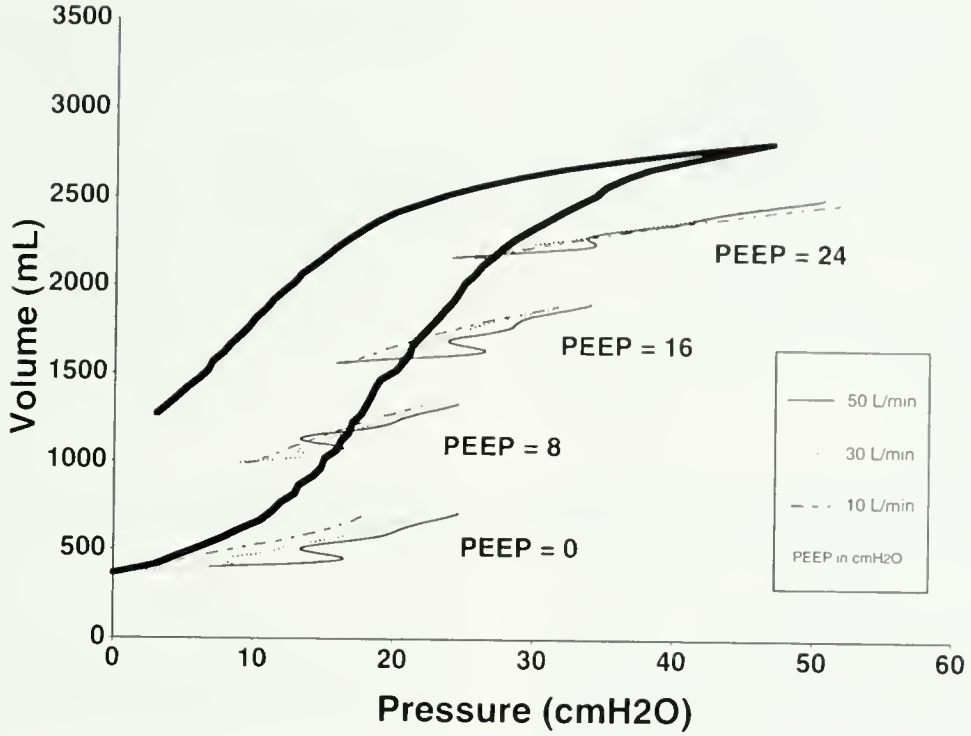

Fig. 5. Subject 4: Note the secretion effect at the high flow of 50 L/min and the failure of the end-expiratory dynamic pressure-volume point to migrate vertically as positive end-expiratory pressure (PEEP) is raised.

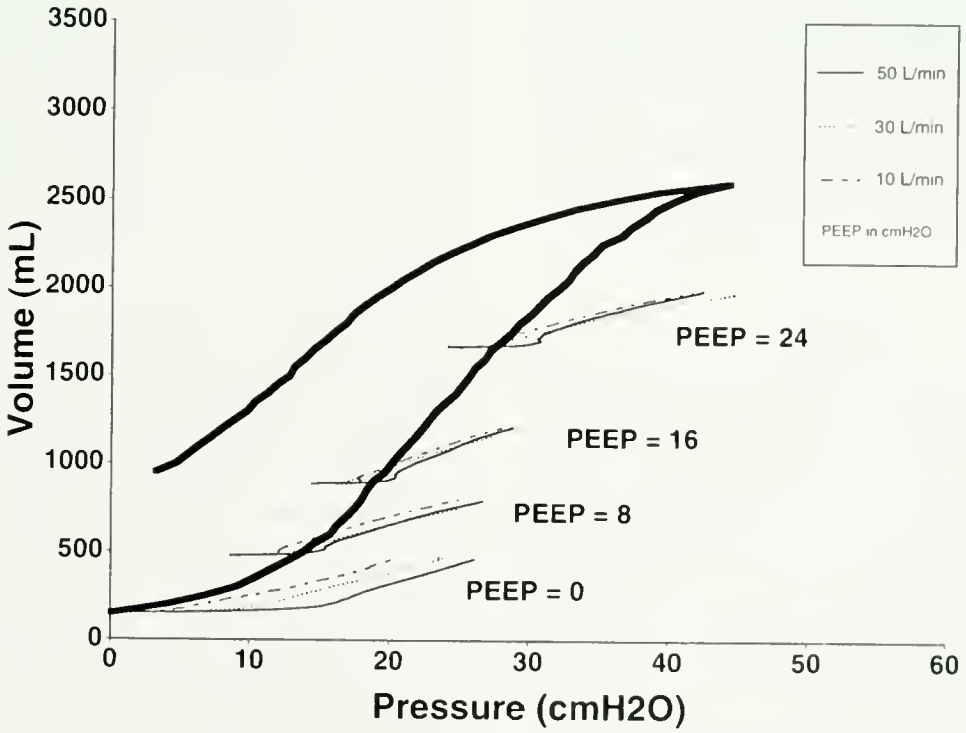

Fig. 6. Subject 5: The rising phases of the dynamic pressure-volume curves are, again, parallel to each other and not parallel to the inspiratory static pressure-volume curve. The end-expiratory dynamic pressure-volume curve is, again, elevated vertically in reference to the static pressure-volume curve. PEEP = positive end-expiratory pressure.

Table 2. Mean Compliance and Lower Inflection Point Measurements for the Static and Dynamic Pressure Curves

		Compliance (mL/cm H_2O)	LIP (cm H_2O)			
SPV		90.6 ± 21.0*	13.2 ± 1.6			
	Flow Setting		LIP at PEEP = 0 cm H_2O	LIP at PEEP = 8 cm H_2O	LIP at PEEP = 16 cm H_2O	LIP at PEEP = 24 cm H_2O
DPV	10	20.2 ± 4.3	3.3 ± 0.3	11.1 ± 0.8	16.9 ± 0.7	26.8 ± 1.5
	30	23.6 ± 5.9	9.0 ± 4.3	13.8 ± 2.4	19.0 ± 2.9	27.9 ± 1.0
	50	26.2 ± 6.8	15.2 ± 7.8	19.1 ± 7.4	23.8 ± 5.5	30.9 ± 1.4
	overall	23.4 ± 6.2				

LIP = lower inflection point
SPV = static pressure-volume curve.
DPV = dynamic pressure-volume curve.
PEEP = positive end-expiratory pressure
*The mean compliance for the SPV curve was significantly greater ($p < 0.001$) than the DPV curve mean compliance. Though mean compliance increased with the flow setting, the increase was not significant. LIP for the DPV curve increased with increasing flow settings and the set PEEP levels. LIP for the DPV did not serve as an indicator for LIP of the SPV

For each individual animal, the magnitude of displacement bore no consistent relationship to the level of PEEP. Mean total FRC for the DPV curves exceeded that of the corresponding SPV curve by (values are mean ± SEM) 267 ± 86 mL, 425 ± 129 mL, and 494 ± 129 mL for PEEP of 8, 16, and 24 cm H_2O ($p < 0.05$, paired *t* tests), respectively.

Table 2 shows the mean compliance and LIP values for the DPV and SPV curves. Respiratory system compliance for the SPV was significantly greater than the compliance measurements calculated from the DPV curves (90.6 vs 23.4 mL/cm H_2O, $p < 0.001$). For the DPV compliance measurements there was a trend, which did not reach statistical significance, toward increasing compliance at increasing flow settings. The LIP of the SPV curve (mean = 13.2 cm H_2O) was not associated with the LIP of the DPV curve.

Discussion

The major findings of this study were the differences between the slopes (compliance measurements) and LIP of the SPV and DPV curves. As well, we observed an elevation in end-expiratory lung volume (EELV) occurring during dynamic tidal ventilation above that anticipated from the SPV. Recent studies of a "slow flow method" DPV measurement reported similarity between static and quasi-dynamic curves.[2,3] These studies generated dynamic curves from ZEEP and delivered an extended low flow—not a tidal ventilation condition but a procedure designed to create a pressure-volume curve.

In a study more directly comparable to ours, Ranieri et al found alterations in DPV curves that were dependent on the prevailing dynamic (PEEP) conditions—a finding similar to the present study.[6] Recent studies also reported evidence of alveolar recruitment above the LIP, which supports our findings of increased EELV during tidal ventilation.[6,7]

In this study we observed differences between DPV and SPV curves that were, generally, expected. Directly predictable from the equation of motion, resistive pressures shifted the "inflection point" of the DPV curves to the right in relation to the flow setting. This "inflection point" represents the transition from resistive to elastic pressure during constant flow ventilation and would not be expected to correspond to (or have the same meaning as) the inflection region identified by the "recruitment" LIP of the SPV curve. The slope of the second (linear) portion of the DPV curve was less (flatter) than that of the SPV curve, indicating a lower dynamic compliance. We speculate that additional pressure was needed to distribute V_T, to displace secretions into the alveoli, and to overcome viscoelastic forces during DPV curve generation. Since the ascending limbs of the DPV curves generated by a wide range in flows appear to approximately parallel each other, the difference between SPV and DPV second phase slopes cannot be explained on the basis of viscoelastic force generation alone. We speculate that in this edematous model,[8] considerable pressure must be generated to overcome viscoelastic forces *and* to peripheralize secretions, even when they are not overtly evident, as in Figures 2 and 5.

Our study identified increased EELV during dynamic conditions. As recently described on theoretical grounds,[9] the EELV during dynamic conditions should be significantly greater than predicted by the inspiratory limb of the SPV curve at the same end-expiratory pressure. Provision of tidal ventilation not only helps to arrest absorption atelectasis but also may apply a pressure higher than that

existing at PEEP, perhaps achieving the opening threshold of lung units that remain closed under static conditions at the same end-expiratory pressure.

The SPV curve can also be used to identify a UIP. Unexpectedly, we did not find a consistent identifiable UIP in our dynamic tracings. Overdistention (as indicated by a UIP) may not have occurred over the pressure range we used (V_T of only 350 mL) or the "beak" sign of overdistention was obliterated by distributive and viscoelastic components of the DPV curve.

Several limitations to this study can be clearly identified. As a model of lung injury induced by a blood-borne agent, the injury mimics the initial phase of "secondary" (type II) acute respiratory distress syndrome.[10] Oleic acid injury is a highly recruitable model, in which airway secretions are usually copious.[8] Our results may not be generalizable across all categories of lung injury. In this study we varied PEEP and flow only while V_T and frequency remained unchanged; the extent of dynamic recruitment for a complete range of ventilator strategies was not investigated. Finally, though gas exchange was continually monitored, for safety purposes, by indwelling arterial blood gas analysis, the important correlation between EELV status and oxygenation was not made during this study.

We conclude that the contours of the SPV curve are not reliably reflected by those of the DPV curve in this model of acute lung injury. This study thus indicates that features of the DPV curve should not be used to guide the selection of PEEP/V_T combinations. Furthermore, an increase in end-expiratory lung volume occurs during tidal ventilation that is not reflected by the classical SPV curve.

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Staffing Exercise Sessions in Pulmonary Rehabilitation

Phillip D Hoberty EdD RRT, Carol Y Chiu RRT, and Rebecca J Hoberty RRT

INTRODUCTION: There is no published standard for the ratio of staff to patients during exercise sessions in outpatient pulmonary rehabilitation. This lack of a standard raises concern about patient safety and the ability to monitor patients adequately. OBJECTIVE: Determine a staff-to-patient ratio standard by examining current practice. METHODS: The directors of the 46 outpatient pulmonary rehabilitation programs in Ohio were surveyed. Directors of 40 (87%) programs responded, and the responses from the 36 programs that conducted pulmonary exercise sessions separately from cardiac sessions were analyzed. RESULTS: The maximum number of staff in an exercise session was 2, with a median of 1. The maximum number of patients in a session was 20, with median of 4. Directors were also asked to rank the importance of 8 factors in determining the reported ratio. From highest to lowest importance, the factors were patient safety, severity of pulmonary disease, availability of space or equipment, entry test data, pulmonary diagnosis, presence of cardiac problems, convenience, and other. Sixty percent of programs restricted the number of patients per session based on staff-to-patient ratio. CONCLUSIONS: Statewide practice for staff-to-patient ratio in Ohio was described. The most common staff-to-patient ratio during exercise sessions was 1:4 or less (78%). Exercise staff work with small groups in order to assure patient safety, to deal with patients' health problems, and to provide adequate facilities. A majority of programs restrict the number of patients per session so as to enforce the reported ratio. *Key words: pulmonary rehabilitation, staffing, staff-to-patient ratio.* [Respir Care 2001:46(7):694–697]

Introduction

Pulmonary rehabilitation is a multifaceted treatment program for patients with a variety of chronic lung diseases. It consists of assessment, exercise training, patient and family education, psychosocial/behavioral interventions, and outcomes assessment, and is being used in the treatment of an increasing variety of pulmonary diseases.[1] Exercise training is considered a mandatory component of successful rehabilitation.[2] Outpatient exercise training sessions are usually conducted as small group sessions during which patients use multiple pieces of equipment for upper and lower body strength and endurance training.[3] It has been suggested that the intensity of exercise should be relatively high, somewhere in the range of 70–80% of the maximum work capacity, as determined by an exercise stress test.[4] This combination of group activity, high-intensity exercise, and the use of multiple modes of exercise during exercise sessions for chronically ill patients raises issues about supervising patient activity and maintaining patient safety in the exercise facility.

Cardiac rehabilitation has a published standard for staff-to-patient ratio during Phase II outpatient exercise sessions.[5] The American Association for Cardiovascular and Pulmonary Rehabilitation Guidelines for Cardiac Rehabilitation require a minimum staff-to-patient ratio of 1:5. Program certification guidelines of the American Association for Cardiovascular and Pulmonary Rehabilitation employ this standard as one of several criteria for awarding certification.[6] Pulmonary rehabilitation, however, has no published standard for this ratio. This is in spite of the fact that the population referred to pulmonary rehabilitation has been identified as being older, functionally more compromised, and presenting with more co-morbidity than patients referred to cardiac rehabilitation.[7] Therefore, in this

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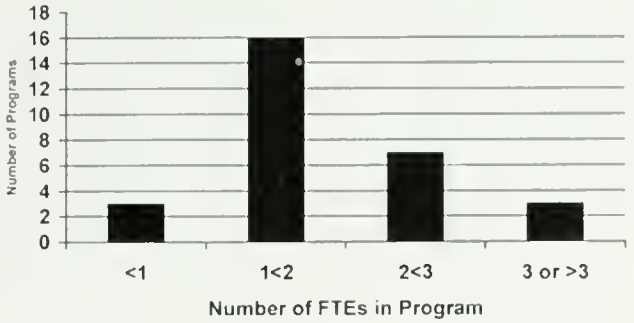

Fig. 1. Number of programs reporting each range of full-time employees (FTEs) ($n = 29$).

compromised population, the lack of standard makes it necessary for each program manager to try to determine an appropriate number of staff and patients for exercise sessions.

The purpose of this study was to describe the staff-to-patient ratio for pulmonary rehabilitation exercise sessions in Ohio. Program managers may consider the ratio, and the criteria on which it is based, in making staffing decisions for their programs and in determining whether or not to increase group size as more patients are referred to rehabilitation.

Methods

We surveyed the program managers of the 46 pulmonary rehabilitation programs in Ohio. The programs consisted of the rehabilitation programs listed for the state in the 1999 American Association for Cardiovascular and Pulmonary Rehabilitation Program Directory and the mailing list of the Rehabilitation Section of the Ohio Society for Respiratory Care.[8] The survey instrument was a written questionnaire consisting of 6 questions (see Appendix). The instrument was designed to gather demographics and to seek answers to 3 fundamental questions:

1. In your outpatient sessions, what is the usual staff-to-patient ratio?

Table 1 Summary of Staff-to-Patient Ratio

	Minimum	Maximum	Mean	Standard Deviation	Median
Staff	1	2	1.313	0.464	1
Patients	1	20	5.486	4.184	4
Ratio*	0.10	1.00	0.296	0.119	0.25

*Staff-to-patient ratio calculated as staff divided by patients

2. On what criteria was the ratio established?
3. Do you restrict the number of patients in a session based on this ratio?

Managers were also asked to report whether patients in pulmonary rehabilitation exercise sessions were separate from those in exercise sessions for cardiac rehabilitation. The questionnaire was pilot tested on 3 directors of local pulmonary rehabilitation programs. The final questionnaire was distributed by mail along with an introductory letter explaining the project. A postage-paid return envelope was included. The questionnaires were coded to identify nonrespondents for follow-up purposes. A follow-up survey was conducted by mail of those who did not respond within 3 weeks of the initial mailing.

Results

Program managers from 40 programs responded with completed questionnaires. This resulted in a return rate of 87%. Because cardiac rehabilitation already has a standard for staff-to-patient ratio, we excluded returns from programs that reported exercising both cardiac and pulmonary rehabilitation patients in the same location at the same time. We analyzed the results of questionnaires from the 36 programs that conducted pulmonary rehabilitation sessions separately from cardiac sessions. Figure 1 describes the number of full-time employee staff for the programs. Sixty-five percent of programs had less than 2 full-time employees. Table 1 summarizes the reported number of staff and patients and staff-to-patient ratio during exercise

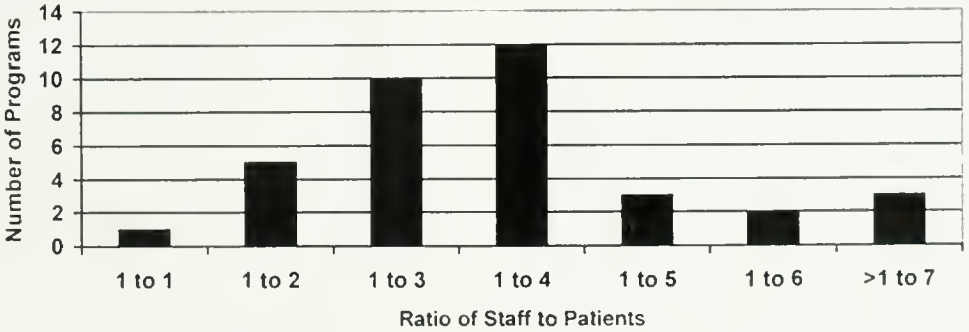

Fig. 2. Number of programs reporting each staff-to-patient ratio ($n = 36$). Ratios are rounded to nearest whole numbers.

Table 2. Ranking of Criteria Used in Establishing Ratio

Rank*	Criteria for Establishing Ratio	Frequency Criteria Was Ranked	Mean Rating†
1	Patient safety	35	2.78
2	Severity of pulmonary disease	35	3.31
3	Availability of space or equipment	35	4.89
4	Pulmonary diagnosis	35	5.42
5	Test data (PFTs, 6-min walk test, etc)	35	5.50
6	Presence of cardiac problems	35	6.36
7	Convenience	32	7.06
8	Other	35	8.47

*Importance in establishing ratio (1 = highest, 2 = second highest, etc) based on mean reported rating
†Missing values (not ranked) made equal to 9 in determining mean rating for all criteria.
PFT = pulmonary function test

sessions. Figure 2 describes the number of programs reporting each staff-to-patient ratio. Seventy-eight percent of programs reported a ratio equal to 1 staff member working with 4 patients or less during the exercise sessions.

Managers were also asked to rank the list of 8 criteria as to their importance in establishing the ratio used in their programs by assigning a rank of 1 for the most important, 2 for the second most important, and so on. The eighth criterion was "other," with mangers asked to list the specific criteria. Table 2 reports the mean ranking for each criterion. In determining the mean, a value of 9 was assigned to the items not ranked by the manager.

An issue that is as important as identifying the ratio standard is whether or not program managers limit the number of patients in a group based on their reported ratios. Sixty percent of managers reported limiting the size of the patient group based on the ratio reported in their programs.

Discussion

Based on the results of the survey research, a statewide practice for staff-to-patient ratio was described for Ohio. Staff members in pulmonary rehabilitation programs generally work with small groups of patients, with a ratio of about 1 staff member for 4 patients. The average number of patients was smaller than that reported in national data about patient group size in pulmonary rehabilitation programs and slightly smaller than certification requirements for cardiac rehabilitation programs.[5,9] The most important criterion reported in establishing the ratio was patient safety. The specific patient safety issue was not determined. The criterion ranked as second was severity of illness, the third-highest ranked criterion was availability of space or equipment, and fourth through sixth related to the patient's diagnoses and test results. Our results suggest that programs use concern for patient safety and disease severity as the primary limiting factors in establishing a ratio, though some programs may limit the size of the exercise group primarily because of a lack of additional space or equipment. Convenience, which ranked seventh, is very low in importance.

The survey was conducted in one state only, so the conclusions can be applied only to that population of programs. The survey instrument did not include "staff availability" as a ranked criterion for establishing the staff-to-patient ratio. Further research on staff-to-patient ratio should determine the specific concerns for patient safety and the role that availability of staff, space, or equipment play in establishing the ratio. Future research should also determine staff-to-patient ratio at the national level.

Based on the results, program managers and medical directors must consider whether increasing the size of patient groups without simultaneously increasing the number of staff substantially decreases patient safety or adequately responds to the disease/severity mix of patients in the group.

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Pulmonary Rehabilitation Staff/Patient Ratio Study

Directions Please answer the following questions concerning the exercise sessions in your pulmonary rehabilitation program. Please answer all the questions unless directed not to

1 Do you have a pulmonary rehabilitation program in which the exercise sessions are separate from other wellness programs, such as cardiac rehabilitation?

_____Yes (Please go on to question #2.)

_____ No (Thank you Please return this questionnaire in the envelope provided)

2 What is the average number of patients who participate in exercise sessions each week? _____
How many FTEs make up your rehabilitation staff? _____

3. In your pulmonary rehabilitation exercise sessions, what is the usual staff-to-patient ratio?

_____ staff to _____ patients

4 On what criteria have you established this ratio? (Please select or list the criteria you used and rank their importance in developing the ratio (1 = most important, 2 = second most important, etc).

CRITERIA	RANK
_____ convenience	_____
_____ pulmonary diagnosis	_____
_____ severity of pulmonary disease	_____
_____ patient safety	_____
_____ test data (PFTs, ABGs, 6-minute walk, etc.)	_____
_____ availability of space and equipment	_____
_____ presence of cardiac problems	_____
other _____	_____

5. Do you restrict the number of patients in an exercise session by enforcing this ratio?

_____ Yes
_____ No

6. Do you have different staff-to-patient ratios for different patient groups you treat (such as one ratio for COPD patients and another ratio for restrictive disease patients or pre-thoracotomy patients)?

_____ YES (Please list the ratios and describe why these are appropriate. Thank you Please return the questionnaire in the envelope provided)

_____
_____

_____ NO (Thank you Please return the questionnaire in the envelope provided)

Effect of Prolonged Low-Dose Methylprednisolone Therapy in Acute Exacerbation of Idiopathic Pulmonary Fibrosis

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We report on a 74-year-old man with an acute exacerbation of idiopathic pulmonary fibrosis (IPF) who was successfully treated with prolonged low-dose methylprednisolone, initiated at a loading dose of 2 mg/kg, followed by 2 mg/kg per day for 14 days. The dose was then tapered. The exacerbation observed on the chest radiograph and high-resolution computed tomography was found to have abated after the treatment. This successful case suggests the feasibility of this methylprednisolone treatment protocol for patients with IPF accompanied by accelerated deterioration. Furthermore, this case suggests possible similarities between acute exacerbation of IPF and late acute respiratory distress syndrome, as the same treatment protocol was previously proved to be beneficial for patients with late acute respiratory distress syndrome. *Key words: idiopathic pulmonary fibrosis, usual interstitial pneumonia, respiratory failure, acute respiratory distress syndrome.* [Respir Care 2001;46(7):698–701]

Introduction

Idiopathic pulmonary fibrosis (IPF), also referred to as cryptogenic fibrosing alveolitis, is a fatal disorder with a reported median survival of 3 to 6 years.[1] Although its clinical course is generally chronic, some IPF patients worsen abruptly. Kondoh et al[2] reported 3 cases of IPF with acute clinical exacerbation. Histology findings from open lung biopsy from these patients showed both usual interstitial pneumonia (UIP) and organizing acute lung injury pattern. Colby et al also noted in some UIP patients a fulminant and usually fatal acute diffuse alveolar damage superimposed on the UIP.[3] Thus, IPF can cause acute deterioration in some patients during its chronic course. However, there have been no studies about treatment for acute IPF exacerbation. Although high-dose corticosteroid therapies, such as 1 g/d of methylprednisolone for 3 days, have been reported,[2,4] consensus has not been reached.

Recently, Meduri et al reported that prolonged administration of low-dose methylprednisolone in patients with unresolving acute respiratory distress syndrome (ARDS) was associated with improvement in lung injury score and reduced mortality.[5] In the present case we used the Meduri et al methylprednisolone treatment protocol with an IPF patient suffering accelerated clinical deterioration, and evaluated efficacy.

Case Summary

A 74-year-old man was referred in July 1999 because of a 6-month history of dyspnea on exertion. No other symptoms were present. The patient was in his usual state of health before the onset of dyspnea. His medical history included cholelithotomy. He was a former smoker. He had not had any inhalation episodes.

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On examination, the patient appeared comfortable. Heart rate was 72 beats/min, respiratory rate 14 breaths/min, and he was afebrile. He had fine, bibasilar, inspiratory "Velcro-like" crackles. Heart and abdomen were within normal limits. There was no peripheral cyanosis or edema, but he did have clubbing of the fingers. Arterial blood gas values on room air were pH 7.41, arterial partial pressure of car-

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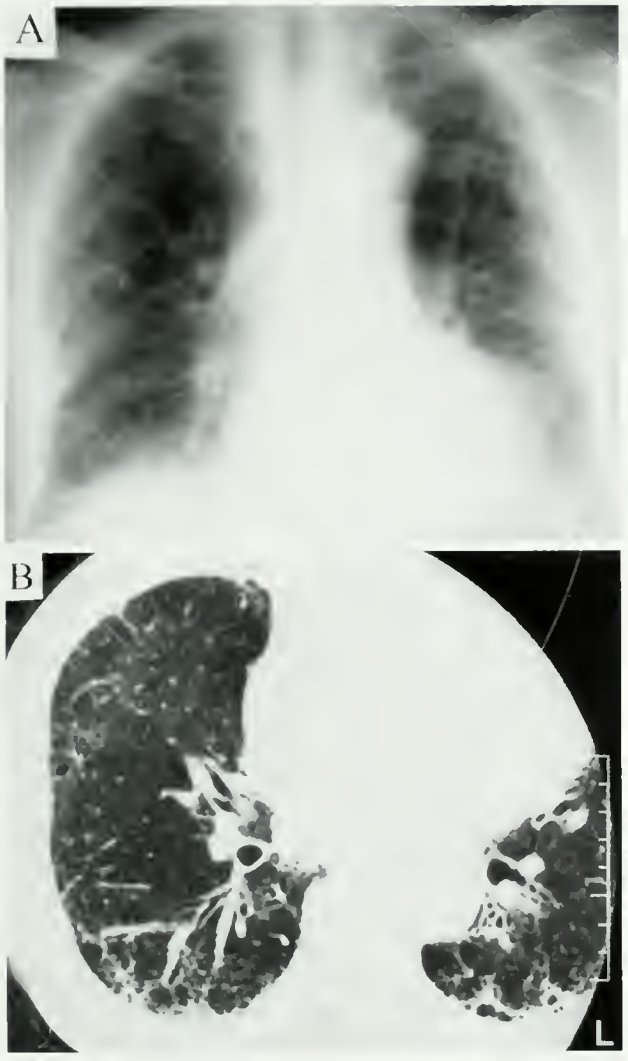

Fig. 1. Chest radiograph (A) and high-resolution computed tomography (HRCT) (B) obtained on admission. The chest radiograph reveals bilateral reticular density and consolidation with predilection for the lower lung zones. HRCT reveals consolidation, intralobular interstitial thickening, and traction bronchiectasis. These abnormalities in HRCT had lower lung zone predilection and predominantly peripheral distribution.

bon dioxide 36 mm Hg, and arterial partial pressure of oxygen (P_{aO_2}) 64 mm Hg. Electrocardiography findings were normal. C-reactive protein level was not elevated. No evidence was found of connective tissue disorder. Figure 1 shows the initial chest radiograph and high-resolution computed tomography. Pulmonary function test results were: vital capacity 1,860 mL (59.8% of predicted), forced expiratory volume in the first second 1,440 mL (68.5% of predicted), ratio of forced expiratory volume in the first second to forced vital capacity 83.2%, and diffusing capacity of the lung 4.95 mL/min/mm Hg (38.8%).

Bronchoscopy with transbronchial biopsy and bronchoalveolar lavage (BAL) showed nonspecific interstitial inflammation but not a histologic diagnosis. BAL findings were: total cell count 3.3×10^5/mL, macrophages 67.7%, lymphocytes 26.2%, and neutrophils 6.1%. Culture of BAL fluid excluded bacterial infection.

Open lung biopsy was performed on July 23 to make a definitive diagnosis. Biopsy specimens were obtained from right upper and lower lobes (S3 and S8). Microscopically, dense collagenous deposition with interstitial fibrosis, infiltrate of lymphocytes and plasma cells, and fibroblastic proliferation were observed in S3. Section S8 showed diffuse dense fibrosis with loss of alveolar architecture, showing honeycomb fibrosis. In both S3 and S8, temporal heterogeneity of fibrosis was observed, characteristic of UIP.[6]

Two weeks after open lung biopsy, low grade fever (> 37 °C) and dyspnea appeared. Chest radiograph and high-resolution computed tomography showed deterioration (Fig. 2). His respiratory status worsened rapidly despite oral administration of levofloxacin. He was intubated on August 12, as we could not keep him adequately oxygenated. Soon after intubation, P_{aO_2}/F_{IO_2} (ratio of P_{aO_2} to fraction of inspired oxygen) was 109, indicating an ARDS state.[7] Swan-Ganz catheterization found wedge pressure to be in the normal range. A ventilator management procedure was designed, employing low tidal volume, transalveolar pressure limitation, pressure-limited ventilation, and permissive hypercapnia.[8-10] Facile BAL was performed after intubation and the culture was negative. Methylprednisolone therapy was then initiated at a loading dose of 2 mg/kg, followed by 2 mg/kg per day for 14 days.[5] The dose of methylprednisolone was then tapered. One fourth of the total daily methylprednisolone dose was given every 6 hours, until methylprednisolone was changed to oral prednisolone. Cefozopran 2 g (a third-generation cephalosporin available in Japan) and erythromycin 1 g/d were administered intravenously in consideration of possible infection. BAL was performed again on Day 5 of mechanical ventilation (total cell count 1.4×10^5/mL, macrophages 39.3%, lymphocytes 21.4%, neutrophils 35.7%, eosinophil 3.6%) and ventilator-associated pneumonia was excluded. The patient's condition and respiratory status gradually improved, without any severe complications, and he was extubated on August 23. Intravenous methylprednisolone was changed to an oral maintenance dose of prednisolone 20 mg on alternate days and ciclosporin 150 mg per day as a maintenance therapy (Fig. 3).

Infection by viruses, mycoplasma, chlamydia, and legionella were all excluded by serological studies throughout the course.

Three months after the exacerbation, chest radiograph and high-resolution computed tomography findings were improved (Fig. 4) and the patient was discharged on November 8. The patient has been under observation for at least 6 months after discharge and has not deteriorated.

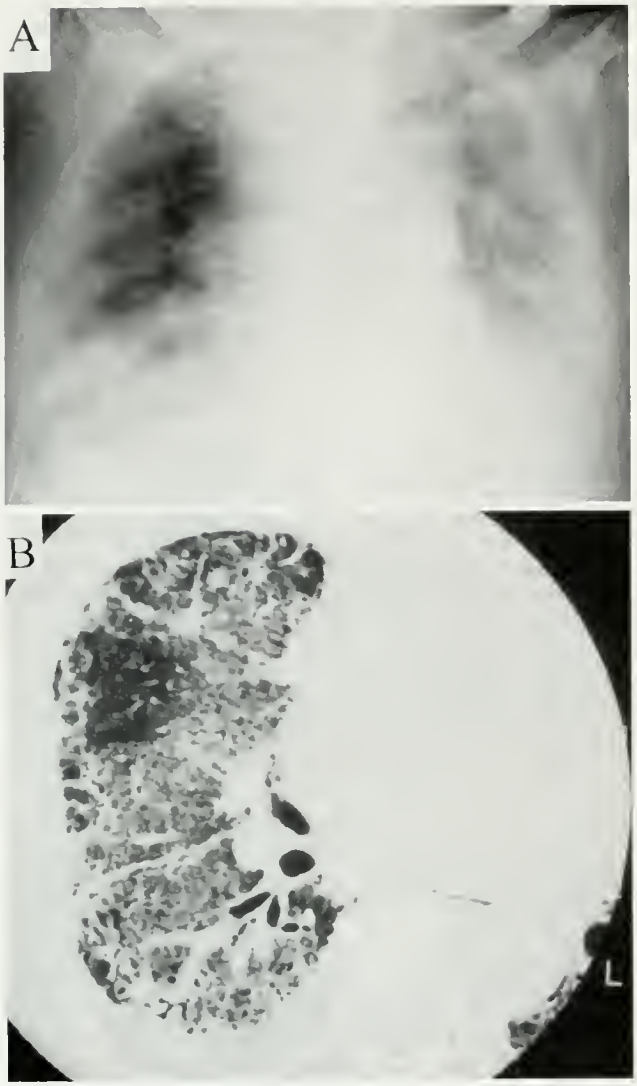

Fig. 2. Chest radiograph (A) and high-resolution computed tomography (HRCT) (B) at acute exacerbation, showing increased bilateral infiltrates. The HRCT shows ground-glass opacity with diffuse distribution.

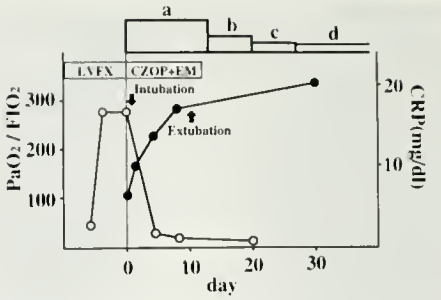

Fig. 3. Clinical course and change in the ratio of arterial partial pressure of oxygen to fraction of inspired oxygen (P_{aO_2}/F_{IO_2}) (closed circles) and C-reactive protein (CRP) (open circles). Vertical line marks August 12 (Day 0), when corticosteroid treatment was initiated. Squares at the top denote the corticosteroid therapy. a: 2 mg/kg of methylprednisolone per day, with a loading dose of 2 mg/kg on Day 0. b: 1 mg/kg of methylprednisolone per day. c: 0.5 mg/kg of prednisolone. d: 20 mg of prednisolone on alternate days, with 150 mg of cyclosporin per day. LVFX = levofloxacin. CZOP = Cefozopran. EM = erythromycin.

Discussion

We described a patient with IPF accompanied by accelerated deterioration, who was successfully treated with prolonged low-dose methylprednisolone. Kondoh et al proposed a definition of acute exacerbation of IPF as follows: (a) exacerbation of dyspnea within a few weeks, (b) newly developing diffuse pulmonary infiltrates, (c) deterioration of hypoxemia ($P_{aO_2}/F_{IO_2} < 225$), and (d) absence of apparent infectious agents.[2] Our patient met those criteria. Although the entity of acute exacerbation of IPF has already been reported,[2] and corticosteroid therapy is likely to be the sole beneficial treatment, the appropriate dose or duration of steroid therapy has not been established. In the absence of trials evaluating the dose or duration of steroid treatment, high-dose corticosteroid therapies, such as 1 g/d of methylprednisolone for 3 days, were frequently used.[2,4]

As for ARDS, there is evidence that corticosteroids are beneficial in the fibroproliferative phase, or late phase, of ARDS.[5,11] The treatment protocol for prolonged low-dose methylprednisolone that Meduri et al recently proposed to treat unresolving ARDS[5] is a loading dose of 2 mg/kg, followed by 2 mg/kg/d from Day 1 to Day 14, 1 mg/kg/d from Day 15 to Day 21, 0.5 mg/kg/d from Day 22 to Day 28, 0.25 mg/kg/d on Days 29 and 30, and 0.125 mg/kg/d on Day 31 and 32. One fourth of the total daily methylprednisolone dose is given every 6 hours. When oral intake is restored, methylprednisolone is changed to a single oral dose. Because the high-dose corticosteroid therapies used in previous reports about acute exacerbation of IPF can easily cause more severe complications such as infection, mineral imbalance, myopathy, and psychosis, and usually fail to withdraw, we tried a modified version of the Meduri et al protocol in this case of acute exacerbation of IPF. We successfully treated the patient, without severe complications, which suggests that the protocol might be useful in IPF exacerbations.

IPF patients can deteriorate for various reasons.[12] Since there was no lung biopsy following the acute deterioration, we are not certain why the patient deteriorated or why he subsequently recovered to his baseline state. However, infectious causes for the deterioration were excluded, so the patient met the criteria of acute exacerbation of IPF proposed by Kondoh et al.[2] It was noted that both UIP and organizing diffuse alveolar damage patterns were observed histologically in patients with acute exacerbations of IPF.[2] Therefore we think that in the present case organizing diffuse alveolar damage was the main histologic feature of

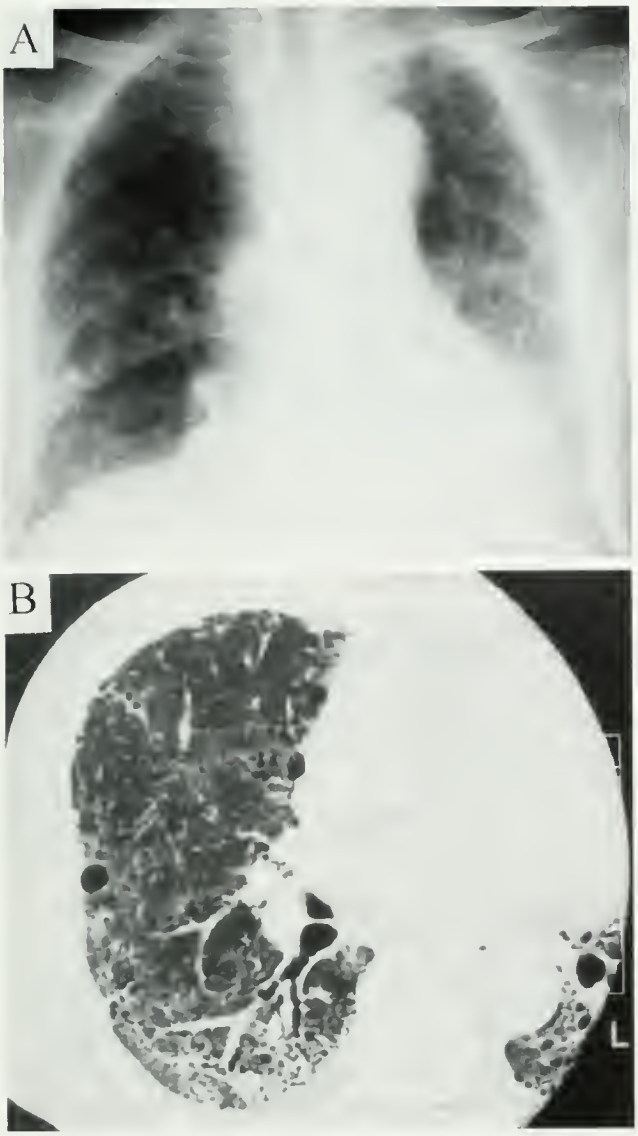

Fig. 4. Chest radiograph (A) and high-resolution computed tomography (HRCT) (B) 3 months after exacerbation. The abnormalities observed at acute exacerbation have improved. The ground-glass opacity on the HRCT had almost disappeared.

the exacerbation, superimposed on the findings of UIP. Considering that histology findings of late ARDS are consistent with organizing diffuse alveolar damage,[13] we hypothesize that the main histologic features are parallel in these 2 clinical conditions. We also hypothesize that the prolonged low-dose corticosteroid treatment reported to be beneficial for patients with late ARDS would also benefit patients suffering acute exacerbations of IPF.[5] Although further studies are needed, the present successful case supports that hypothesis.

In conclusion, we present a patient with acute exacerbation of IPF successfully treated by prolonged low-dose methylprednisolone. This treatment protocol can be substituted for the high-dose methylprednisolone therapies reported previously. Furthermore, there may be parallels between acute exacerbation of IPF and late ARDS and their responses to corticosteroid therapy.

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Co-Existing Granular Cell Tumor and Adenocarcinoma of the Lung: A Case Report and Review of the Literature

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Granular cell tumor (GCT) of the lung is a rare tumor, constituting less than 10% of all GCTs. It is a multicentric tumor and infiltrates into adjoining tissue, but malignant GCT of the lung has not been reported. Diagnosis is usually obtained with bronchoscopic biopsy. Treatment options include bronchoscopic extirpation, laser therapy, and sleeve resection. We present a case of GCT co-existing with adenocarcinoma of the lung and review the literature. [Respir Care 2001;46(7):702–704]

Introduction

Granular cell tumor (GCT) can occur in virtually any location, the commonest area being the head and neck. Usually GCTs are benign, but malignant ones have been reported. GCT of the lung is a rare neoplasm. It was thought to be of myoblastic origin, but recent consensus is that the tumor originates from Schwann cells. The main morphologic feature is the granularity of the cytoplasm. Pulmonary complications from the tumor are due to airway obstruction. Therapeutic options depend on the size and number of the lesions and the extent of lung damage.

Case Summary

The patient is a 35-year-old African-American female with a history of adult polycystic kidney disease, cerebral artery aneurysm, and emphysema. She presented with 2-week history of "flu-like" symptoms and chest tightness. She had an 18 pounds weight loss over the past 3 years. The patient was a 40-pack-year smoker. There was no history of fever, chills, night sweats, hemoptysis or calf pain. She was allergic to penicillin and codeine. Her occupational history was unremarkable.

Physical examination showed an alert but emaciated and malnourished young woman. She was afebrile and her vital signs were stable. There was no evidence of skin lesions, lymphadenopathy, or increased jugular venous pressure. Chest examination showed fair air entry and few crackles bilaterally, which cleared with coughing. There were no rhonchi, wheezes, stridor, or other adventitious sounds. Breast tissue was soft, with no palpable masses. Heart rhythm was regular, with no murmurs or rub. Liver span was 8 cm. There was no evidence of edema, clubbing, or cyanosis.

Laboratory data showed hemoglobin of 11.7 gm/dL, white blood cell count of 5,400/mL, and normal serum chemistry except for a bicarbonate level of 31. Chest radiograph showed hyperaerated lung fields and a 2 cm nodule in the left upper lobe. Computerized tomography confirmed the presence of a 2 cm nodule in the left upper lobe, in addition to diffuse emphysematous changes in lung parenchyma. There was no evidence of hilar or mediastinal lymphadenopathy.

Pulmonary function tests showed a severe obstructive ventilatory defect, with air trapping and severely reduced carbon monoxide diffusing capacity.

The patient underwent a flexible bronchoscopy, which revealed a 0.5 cm lesion at the major carina, a 1 cm polypoid mass in the right upper lobe, and no endobronchial lesion in the left upper lobe or other segments. Biopsies were taken from the carinal and right upper lobe lesions. Bronchoalveolar lavage (BAL) fluid from both lungs showed spindle cells with abundant granular eosinophilic cytoplasm and spindle-shaped nuclei with finely granular chromatin and small nucleoli (Fig.1). These cells stained strongly with S-100 protein, consistent with a diagnosis of GCT, with no evidence of malignancy.

Taking into account her decreased pulmonary function reserve and increased risk of complications, including possible chronic ventilator dependence, the patient underwent

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Fig. 1. Section from carinal biopsy (magnification 20, S-100 stain) shows a submucosal infiltrate of cells (arrow) with small bland nuclei and abundant granular eosinophilic cytoplasm, and the cells stain strongly with the S-100 protein.

a wedge resection of the left upper lobe nodule. This revealed moderately differentiated adenocarcinoma with bronchoalveolar features. She did not receive any radiation or chemotherapy. As the patient did not have any symptoms due to the GCT, and because of the diffuse involvement, she did not receive any specific therapy and was scheduled for close follow-up.

Discussion

GCT was originally described by Abrikossoff[1] in 1926, and the first pulmonary case was described in 1939 by Kramer.[2] GCT is rare and the exact incidence of the tumor overall is not known. In one large series of 110 patients with GCTs in a 32-year period at 2 centers, the tumors accounted for 0.03% of more than 410,000 surgical specimens over the same period of time.[3] The commonest area of involvement is the head and neck, specifically the tongue, which constitutes 23–28% of all GCTs.[3] Other sites include mucous membranes of the gastrointestinal tract (particularly the esophagus) and the female genital tract.[3] Pulmonary GCTs account for 6–10% of all GCTs.[4] Most pulmonary GCT lesions are in the lower respiratory tract and form polypoid and endobronchial masses.[4] Rarely they can occur in the posterior mediastinum.[5]

Pulmonary GCT can be multicentric and up to 25% can be multiple.[6] They tend to occur in large bronchi, at the sites of bifurcation. This, however, does not indicate malignant potential, though it can mimic pulmonary metastases in patients with other malignancies. Metastatic malignant GCT to the lung forms parenchymal nodules rather than polypoid endobronchial lesions. Patients with familial multiple GCT have been reported to develop malignant GCT with lung metastases.[7] No primary malignant GCT of the lung has been reported, although 3–6% of extrapulmonary GCT lesions are malignant.

The median age for the occurrence of GCT is the fourth or fifth decade, but the incidence ranges from 5 to 59 years of age.[5] GCT occurs equally frequently in men and women.[8] In some studies there was a higher predilection in the African-American population.[5]

A unique form of GCT consisting of alveolar septal infiltration was observed in a 29-week-old male fetus with congenital GCT and systemic involvement.[8]

In 50% of cases, symptoms are of endobronchial obstruction: localized wheezing, chest pain, and cough. Other symptoms are hemoptysis and weight loss. GCT can present as post-obstructive pneumonia. The rest are discovered incidentally at bronchoscopy or surgery for some other condition or on routine chest radiograph. The last usually reveals persistent infiltrates or atelectasis, but solitary pulmonary nodules have been reported.[9]

Pulmonary GCT has an infiltrative character.[6] In one series of patients from Walter Reed Army Medical Center, 40% of the tumors had peribronchial tissue extension. Another interesting feature is thickening of the subepithelial basement membrane, seen in less than 40% of the cases. Recognition of this feature may lead to a search for granular cells and to considering the possibility of GCT.

Macroscopically, the tumor is whitish or pale yellow, and it may appear well circumscribed but in fact is not sharply demarcated and does not shell out readily. Microscopically, GCT tumor cells are arranged in slender bands of connective tissue. The periphery of the tumor is not sharply defined, and detached groups of neoplastic cells may extend for some distance, creating an appearance of infiltration. Individually, the cells are ovoid or polygonal, with abundant eosinophilic, coarsely granular cytoplasm. The nuclei are hyperchromatic but uniform in size and shape. The cytoplasmic granules of GCT tumors are periodic acid-Schiff stain positive, diastase resistant, and consistently stain positive with Luxol fast blue stain.[10]

The immunohistochemistry of the tumor gives clues to its origin. Presently the consensus is that the tumor originates from Schwann cells. The tumor invariably expresses S-100 protein. Most GCTs are also positive for vimentin. Staining for collagen IV and laminin is frequently seen around tumor nests.

Histologic differential diagnosis includes oncocytic carcinoid, metastases of the granular cell variant of renal cell carcinoma, squamous cell carcinoma, signet ring carcinoma, and alveolar soft part sarcoma. These lesions should be differentiated on the basis of immunohistochemistry and morphology.

GCT can occur with other neoplasms, the most common being lung cancer.[11–14] Only 2 other cases co-exist with adenocarcinoma.[12,13] Other associations have been with esophageal and renal carcinomas.[6] Co-existing non-neo-

plastic diseases that have been reported include sarcoidosis, human immunodeficiency virus, pulmonary chondroid hamartoma, and cerebral arteriovenous malformation,[6,15,16] but these are probably coincidental. Hypercalcemia has been reported in 2 patients.[5]

The diagnosis is generally based on bronchoscopic biopsy specimens, but recognition on bronchial brushing or washing has been reported.[17,18]

Pulmonary GCT behaves in a benign manner. No malignant pulmonary GCTs have been reported, although 3–6% of extrapulmonary GCT lesions are malignant. The growth rate of pulmonary tumors is extremely slow. Drake et al reported a recurrence in one patient after 7 years and lack of recurrence in another patient after 12 years, despite a positive marginal pneumonectomy.[6] Spontaneous regression has been reported at other sites, but not in the lung.[19]

Therapeutic options need to be individualized. Patients with asymptomatic lesions can be followed without therapy.[20] Conservative approaches include bronchoscopic extirpation, laser therapy, or sleeve resection.[21–23] These methods are preferable if the distal lung parenchyma is preserved or if the tumors are multifocal.[23] Daniel et al proposed that tumors smaller than 8 mm are amenable to endoscopic removal.[22] In cases with extensive lung injury from pneumonia or bronchiectasis, a lobectomy or pneumonectomy may be needed.

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The Future of Respiratory Care

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Respiratory Care: The Journal
Conclusions

The term respiratory care has more than one meaning, referring both to a subject area within clinical medicine and to a distinct health care profession. In the light of several fundamental transformations of health care during the 20th century, this article reviews the history of respiratory care in both of these contexts and offers 10 predictions for the future: (1) Less focus on raising P_{aO_2} as a primary goal in managing patients with acute hypoxemic respiratory failure. (2) More attention to the adequacy of tissue oxygenation in such patients, irrespective of P_{aO_2}, and the emergence of "permissive hypoxemia," analogous to permissive hypercapnia, in managing them. (3) Smarter monitors that display information less but process it more, while interacting directly with ventilators and other devices to modify therapeutic interventions. (4) Increased use of and expertise with noninvasive ventilation, with a corresponding decrease in intubations and complications, in treating patients with acute exacerbations of COPD. (5) Increased use of triage in the intensive care unit, including earlier determination of the appropriateness of maximal supportive intervention. (6) Greater use of protocols in patient assessment and management, in all clinical settings. (7) Increased awareness of, expertise in, and resources for palliative care, with a more active and acknowledged role for respiratory therapists. (8) Accelerating progress in smoking cessation and prevention, and also in early detection and intervention in COPD, led by the respiratory care profession. (9) An increasing presence and impact of respiratory therapists as coordinators and care givers in home care. (10) A continued and enlarging role for the journal Respiratory Care in disseminating research findings, clinical practice guidelines, protocols, and practical educational materials in all areas of the field. [Respir Care 2001;46(7):705–718]

Introduction

Whether one reckons the beginning of the new millennium as the year 2000 or the year 2001, we are at an auspicious time in human history, and no aspect of our current civilization is more important to that history than health care. Having been accorded the honor of presenting the Louis Mark Memorial Lecture at the American College of Chest Physicians' annual convention for the year 2000, I have chosen to discuss the facet of health care that is of greatest concern to the readers of this journal: *respiratory care*.

I will begin by discussing what is meant by *respiratory care*, as it is a term with different meanings in different contexts. I will review the events and developments that have shaped respiratory care until now, so that we can understand why it exists as it does and also pose some rational thoughts about what is likely to happen in the future. In the process, I will make 10 specific predictions of what we are likely to see in the coming decades.

The term *respiratory care* has 3 distinct meanings.[1] First, it is a subject area within science, technology, and health care. It is, second, a distinct health care profession. And, third, it is the name of the peer-reviewed journal in which these words appear. I will discuss the history and future of each of these 3 meanings of *respiratory care* and share some personal perspective developed during nearly 30 years of work in this field.

Respiratory Care: The Subject Area

Definition and Characteristics

When I arrived at the University of Colorado in 1971 as a medical resident, Tom Petty had just published his landmark book, *Intensive and Rehabilitative Respiratory Care*,[2] in which he wrote:

> The development of an organized team approach for the management of patients with acute respiratory failure has been a major advance of today's medicine, and has provided the arena for systematic, physiologically oriented care. The disciplines of internal medicine, surgery, anesthesiology, nursing care, inhalation therapy, and physical medicine and rehabilitation all come to bear on problems presented by each individual case.[2]

As in so many areas of pulmonary medicine, Dr Petty got it right the first time and was many years ahead of his time. Thus, respiratory care as a subject area is a collection of principles, skills, and patient needs that have arisen because of the nature of respiratory disorders and their effects on patients.

Incorporated in Petty's original definitional statement are 4 key characteristics: respiratory care is *systematic*, it is *based on physiology*, it is *multidisciplinary*, and it is *defined by the needs of patients*. I will emphasize these 4 characteristics herein because they are at the very core of the subject area respiratory care as we know it today.

Respiratory care includes portions of several other fields, but it is not the same as any of them. It incorporates aspects of critical care medicine, pulmonology, anesthesiology, nursing, physical therapy, and clinical engineering. Its components are numerous and include pulmonary function testing, arterial puncture and blood gas analysis, endotracheal intubation and all aspects of airway management, mechanical ventilation both invasive and noninvasive and associated functions, the management of respiratory tract secretions, and the administration of aerosols. Also prominently on the list are chest physical therapy, pulmonary rehabilitation, and home care.

Transformations in Health Care During the 20th Century

Health care changed more in the century just concluded than ever before in human history. Although the changes include previously unimaginable medical wonders, not all the changes were for the better. A great internist, Paul Beeson, who lives in Seattle and is now in his 90s, played a major role in the achievements of internal medicine in the 20th century. For a special millennium issue of the *Annals of Internal Medicine*, Beeson was interviewed about the changes he had witnessed in medicine,[3,4] and he cited 3 major transitions or transformations in medicine in the 20th century.

The first of these was the transition of health care from the home and doctor's office to the hospital setting, which took place following the Second World War. The second transformation was the explosion of knowledge and technology that accompanied the growth of academic hospitals and medical schools, along with an enormous expansion of their facilities and faculties, starting in about 1950 and continuing into the mid-1980s. We are contending today

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Table 1 History of Mechanical Ventilation in the Critically Ill

Period	Ventilator Characteristics	Management Targets and Published Literature
Before 1970	Simplicity Limited capability Empiricism	Bedside observation (Blood gases not yet widely available) Anecdotal reports and descriptions
1970s to early 1980s	New modes Increasing capability and complexity	Blood gases Airway pressures Immediate physiologic changes Anecdotal clinical series
Mid-1980s to early 1990s	Microprocessor control New and combined modes More complexity	Short-term changes Complications Small-scale clinical trials
Last 10 years	New targets Brand-specific modes Quest for simplicity	Outcomes Costs Large-scale clinical trials

with the third 20th-century transformation in health care, which Beeson calls the industrialization of medicine.[3] This has shifted control of health care, and of what its practitioners do, from those who deliver health care, to administrators and business people, from whom the clinicians now take orders.

These fundamental transformations of health care in general are echoed in the history of respiratory care. To bring this home I will take as an example the history of mechanical ventilation of the critically ill, and especially of patients suffering acute hypoxic respiratory failure.

History of Mechanical Ventilation for Acute Respiratory Failure

Before about 1970, mechanical ventilation was crude by today's standards, but straightforward (Table 1). Ventilators that could deliver a predetermined tidal volume (V_T) with each breath were replacing earlier machines that could only apply a pre-selected pressure to the airway, but maximum pressures and flows were limited and monitoring was fairly rudimentary. Arterial blood gas (ABG) measurement was a research tool, and clinicians mainly used bedside physical assessment to gauge the adequacy of ventilatory support. Hospitals did not yet have intensive care units (ICUs). The literature on mechanical ventilation consisted mainly of physiologic studies of animals, observations from the operating room, and anecdotal clinical reports.

The advent of intensive care in the United States in the early 1970s was accompanied by new modes and approaches to ventilatory support, notably volume assist/control and intermittent mandatory ventilation. New ventilators such as the Siemens Servo 900 series offered new modes, along with increased pressure and flow capabilities—but also greater complexity for the user. Textbooks and articles dealing with mechanical ventilation proliferated dramatically. These publications focused on airway pressures, ABG measurements, and immediate physiologic changes in response to alteration of ventilator settings. The clinical literature consisted of anecdotal series. Although investigators postulated more effective gas exchange with the new features, any influence of ventilator modes and settings on actual patient outcomes was conjectural if thought about at all.

By the late 1980s, there were new and combined modes, and pressure-targeted ventilation had returned to compete with (and sometimes to combine with) volume-pre-set ventilation. ICU ventilators were microprocessor-driven and ever more complex—not only in their innards but also in what the clinician had to master in order to use them effectively in managing patients.[5] Terminology related to modes of ventilation became more confusing as ventilator manufacturers offered their own variations and combinations of common basic features, each with its own name, apparent uniqueness, and claimed exclusivity.[6,7]

During the 1980s most of the literature still dealt with short-term changes such as alterations in arterial oxygenation, compliance, and airway pressures in response to changes in mode or other manipulations. At about this time the attention of clinicians was also increasingly drawn to the adverse effects of mechanical ventilation, and especially to ventilator-induced lung injury, which were apparently the result of the increased capabilities of the new machines or at least how clinicians were using them. Starting around 1990, new targets and goals for ventilatory support began to emerge for patients with acute respiratory failure, which were intended to prevent or reduce the severity of ventilator-induced lung injury. Published results

Table 2. Elements in the Management of Acute Respiratory Distress Syndrome

1. Oxygenation of the arterial blood
2. Support of ventilation
3. Treatment of the primary condition precipitating acute respiratory distress syndrome
4. Monitoring the patient, the ventilator, and other aspects of management
5. Prevention, recognition, and treatment of complications

from small-scale clinical trials of mechanical ventilation targeted at these new goals began to appear.

In the last few years, mechanical ventilation has, in a sense, come full circle, to a renewed quest for simplicity in management. This is not because the machinery and our knowledge are rudimentary, as was the case 30 years ago, but because we are realizing that we may not have been looking at the right end points, and because what we have been doing costs too much. Studies being published today focus on outcomes and costs, and we now have available, for the first time, data from large-scale randomized clinical trials examining at least some aspects of mechanical ventilation.

Managing Acute Respiratory Distress Syndrome

I would like to take the management of acute respiratory distress syndrome (ARDS) as my case in point in illustrating the trends I have just summarized, and to use it as the springboard for predicting what is likely to happen in respiratory care in the larger sense. Management of ARDS patients basically consists of 5 tasks, as listed in Table 2. We oxygenate the arterial blood, traditionally using arterial partial pressure of oxygen (P_{aO_2}) as the measure of that. We support alveolar ventilation, using arterial partial pressure of carbon dioxide (P_{aCO_2}) and pH as the indices. We treat the primary problem insofar as we are able, and here I refer not to treating ARDS itself but the precipitating process that led to development of the syndrome, such as a specific infection producing sepsis. Fourth, we extensively monitor both the patient and the tools that we are using to manage the patient. And, finally, we do our best to prevent, to recognize early, and to treat complications that arise.

This list describes in a nutshell what is involved in managing ARDS. To provide a framework for speculating about the future of respiratory care in this area, I would like to examine the first 2 elements on the list—that is, the things that we do in management of ARDS that are targeted at supporting gas exchange. I will discuss ventilation first, because we have come further in the evolution of our thinking, and more scientific evidence from ARDS patients is available in this area than with oxygenation.

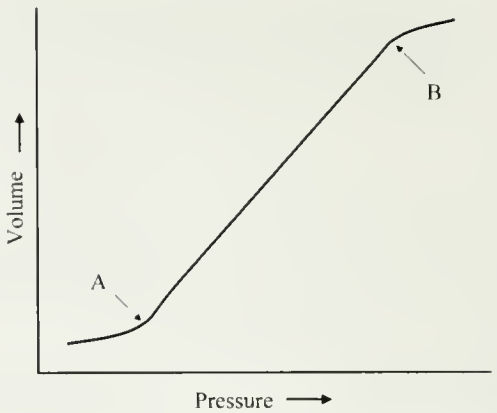

Fig. 1. Idealized pressure-volume curve of the respiratory system of an acute respiratory distress syndrome patient, as measured during a very slow inspiration in the absence of muscular activity. The curve reflects lung and chest-wall compliance (ie, the change in volume for a given change in pressure). Point A, the lower inflection point, indicates increased compliance accompanying recruitment of collapsed alveoli. At pressures and volumes above Point B, the upper inflection (or deflection) point, compliance is reduced because of alveolar over-distention.

Managing Ventilation: Lung Protection and Permissive Hypercapnia

Traditionally, a main goal of mechanical ventilation was to "normalize" gas exchange. That is, we sought to eliminate or minimize evidence of gas exchange impairment, to get as close as possible to a normal P_{aO_2}, right-to-left shunt, venous admixture, or ratio of P_{aO_2} to fraction of inspired oxygen (P_{aO_2}/F_{IO_2}), and also to keep P_{aCO_2} and pH within normal limits. These values are quite natural as goals and it is no wonder that they were pursued for the first 25 years or so after ARDS was first described. However, in the 1990s the management and support of alveolar ventilation and arterial oxygenation has begun to be viewed in a different light and, as a result, the goals and strategies of managing ventilation have changed.[8]

This shift in management is due mainly to a heightened awareness of the alveolar damage that can be done to an ARDS patient if the lung is over-distended during inspiration and insufficiently held open at end-expiration. Figure 1 shows an idealized depiction of the pressure-volume relationship of the respiratory system of an ARDS patient and illustrates the rationale for the new ventilation strategy.

As distending pressure is progressively applied, beginning at a low lung volume, little lung expansion initially occurs because much of the alveolar space is collapsed or consolidated. With additional pressure, recruitable alveoli open and lung compliance increases. However, as inflating pressure is further increased, some alveoli become over-distended and overall respiratory system compliance begins to decrease. The point on the pressure-volume curve

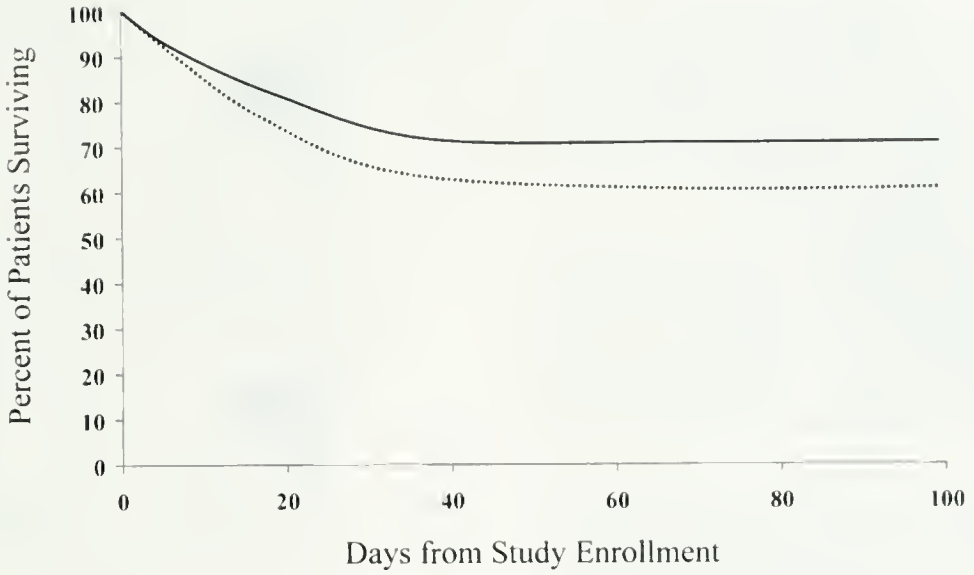

Fig. 2. A smoothed depiction of overall survival among acute respiratory distress syndrome (ARDS) patients managed according to a lung-protective ventilatory strategy (tidal volume 6 mL/kg, solid line) versus traditional ventilation (tidal volume 12 mL/kg, dashed line), using data from the National Institutes of Health-sponsored ARDS Network study. (Adapted from Reference 14.)

at which the lung opens and compliance increases is called the lower inflection point (point A on the figure), and the point at which over-distention occurs and compliance decreases is called the upper inflection (or, more properly, deflection) point (point B).

Numerous studies using experimental animals have shown that tidal ventilation that increases peak lung inflation beyond the upper inflection point, or that allows lung volume to fall below the lower inflection point on expiration, damages normal lungs and greatly augments injury in previously damaged lungs.[9,10] It follows that, in order to minimize ventilator-induced lung injury, tidal ventilation ought to take place between these 2 points on the pressure-volume curve.[11]

Figure 1 is consistent with the available evidence both from animals and humans with ARDS. However, it is greatly simplified, and the inflection and deflection points are typically difficult to identify consistently at the bedside in actual patients. Nonetheless, the management of ARDS has evolved to be more consistent with tidal ventilation in the middle part of the curve. This involves limiting peak alveolar distending pressure, primarily by using lower V_T than before.

I need to point out that the relevant pressure is what is applied to the *alveoli*—end-inspiratory plateau pressure—and not what is measured in the ventilator circuit during inspiration. For a whole generation, clinicians were misled into tracking peak airway pressure as a reflection of the risk of barotrauma and ventilator-induced lung injury. Peak pressure reflects resistance to air flow (ie, things proximal to the alveoli) as well as lung-chest wall compliance, whereas plateau pressure assesses only the latter.

The use of smaller V_T in ARDS patients often results in hypercapnia. However, rather than increasing minute ventilation in an attempt to bring down P_{aCO_2}, current management allows hypercapnia (and respiratory acidosis) to persist as an acceptable price to pay in order to protect the lungs from injury. Hence the term *permissive hypercapnia*.

Although early clinical series by Hickling et al[12] and a small single-center clinical trial by Amato et al[13] reported improved survival of ARDS patients managed with lung-protective ventilation and permissive hypercapnia, compelling evidence of the efficacy of this management has only recently been published. I refer to the ARDS Network study, a large-scale multicenter clinical trial sponsored by the National Institutes of Health (NIH).[14] This study enrolled ARDS patients diagnosed by standardized criteria and randomized them to be managed either with the new lung-protective ventilation strategy (corrected V_T of 6 mL/kg or less, and end-inspiratory plateau pressure kept below 30 cm H_2O) or more traditional mechanical ventilation (V_T of 12 mL/kg and plateau pressure of 50 cm H_2O or less). V_T was determined according to calculated ideal body weight, which turned out to be on average about 20% less than the patients' actual admission weights.[14]

Figure 2 depicts the main results of the ARDS Network study in terms of overall survival.[14] As you can see, there was a clear and both clinically and statistically significant difference in outcome with the 2 ventilation management strategies. The mortality rate in the patients receiving traditional ventilatory management was 40%, versus 31% in the patients managed with lung-protective ventilation—22% lower mortality! I think this is the most important

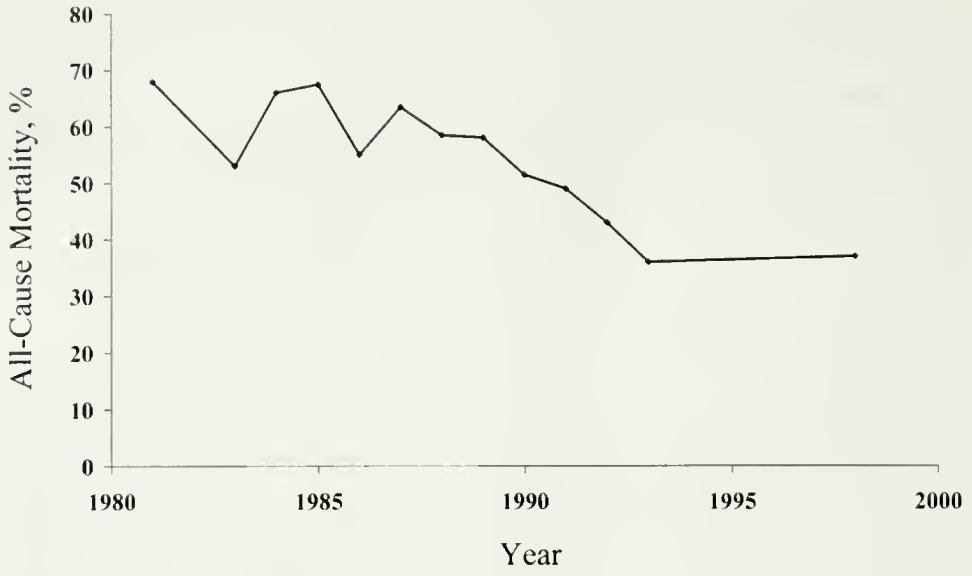

Fig. 3. Unadjusted, all-cause mortality among acute respiratory distress syndrome (ARDS) patients managed at Harborview Medical Center from 1981 through 1998. Diagnostic criteria and precipitating causes of ARDS did not vary significantly during the time period shown. The decrease in mortality is statistically significant and is not substantially altered by adjustment for ARDS risk factors, age, and gender. (Adapted from References 15–18.)

study of mechanical ventilation that has ever been published. It is the first time that it has ever been shown, with a proper, sufficiently powered, controlled clinical trial, that any approach to mechanical ventilation is better than any other in terms of what ultimately happens to the patient.

I need to tell you, though, that even before the ARDS Network study was carried out, some changes were happening with respect to clinical outcomes in ARDS. This is illustrated by the experience of one clinical center, as shown in Figure 3,[15–18] which shows the percentage of ARDS patients who died at Harborview Medical Center from 1981 through 1998. At Harborview, a group of investigators under the direction of Leonard Hudson has been prospectively and consecutively studying every ARDS patient for more than 20 years,[19,20] using a set of diagnostic criteria that are essentially the same as the European-American consensus criteria.[21] During this study period, this group has accumulated what must surely be the largest consecutive series of ARDS cases in the world—well over 2,000 of them.

Figure 3 illustrates an important trend in ARDS mortality. In Ashbaugh and Petty's 1967 report, from Denver, that first described and named ARDS, mortality among their patients was about two thirds.[22] For the next 20 years, reported mortality from Denver and elsewhere among patients who developed this syndrome remained between 60 and 70%.[23] This was the case at Harborview until about 1990. At about that time there began a real and sustained decrease in the mortality of ARDS, as shown in the figure.[23] All-cause mortality declined to about one third, and has remained less than 40% until the present—a decrease of nearly half from the previous mortality rate.[18] This trend is evident not only at Harborview, where half of all ARDS cases occur in patients with multiple trauma, but also at other centers around the world.[24]

I would like to point out that this improvement in ARDS survival was happening *before* we started using lower V_T and other aspects of lung-protective ventilation. Thus, on the one hand we have the ARDS Network study showing that managing ARDS patients with low V_T and limited airway pressures improves survival, and on the other hand we were already doing *something* right, because mortality had already fallen before the new management strategy was introduced.

Managing Oxygenation: Questioning Assumptions and Moving Toward Permissive Hypoxemia

Why do ARDS patients die? It turns out that the great majority of them die from nonrespiratory causes. Figure 4 shows the proportions of deaths among ARDS patients at Harborview that were due to respiratory failure (ie, due to inability to oxygenate the blood and maintain acid-base status) during the same time periods as in the previous figure.[15–18] These pie charts show that most people who get ARDS and subsequently die do not succumb to respiratory failure. The most common cause of death is sepsis and multiple organ failure, which accounted for 31–50% of the deaths during the time period shown. Next was

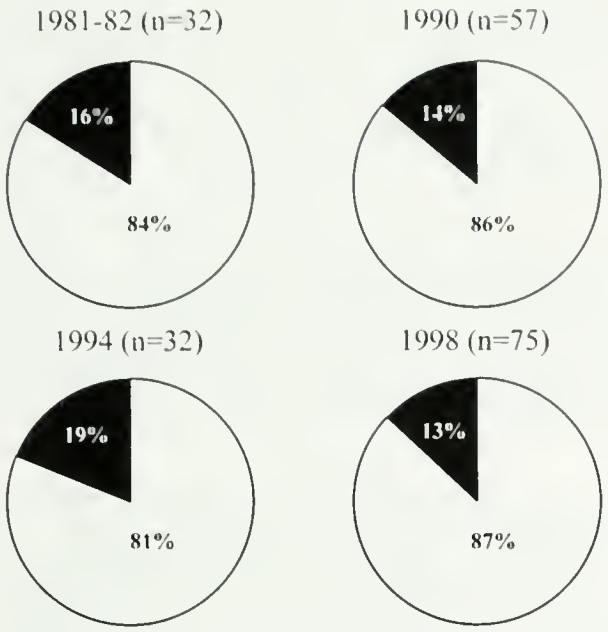

Fig. 4. Causes of death among acute respiratory distress syndrome (ARDS) patients who died at Harborview Medical Center during 4 sampling periods from 1981 to 1998. The proportion of deaths from unsupportable respiratory failure remained between 13 and 19%, while deaths from nonrespiratory causes (mainly from sepsis-multiple organ failure and/or neurologic processes) remained between 81 and 87%. (Adapted from References 15–18.)

cerebral hemorrhage and other forms of central nervous system failure, which caused 19–29% of the deaths. Cardiac, hepatic, hematologic, and other organ failures account for much of the rest. Failure of gas exchange caused only 13–19% of the deaths.

So of all the people who get ARDS and also die, only about 1 in 6 or 1 in 7 dies directly because of respiratory failure. If we consider the fact that the current mortality rate among ARDS patients is about 35%, with only 15% of those dying of gas exchange failure, it means that only 5% of ARDS patients die because of inability to support oxygenation.

I want to deal further with that observation. Of the things we do in managing ARDS patients (see Table 2), number 1 has always been to oxygenate the arterial blood. The assumption has always been that, if we adequately oxygenate the arterial blood, our management has been successful, and that a patient whose oxygenation is maintained has a better chance of survival (Fig. 5). But an increasing body of data suggests that there is reason to question that assumption.

Let's look at the things we do to increase P_{aO_2} in ARDS patients. Number 1 is to increase F_{IO_2}, although for as long as we've been treating ARDS we've been reluctant to increase F_{IO_2} above about 0.5 or 0.6, for fear of oxygen toxicity. The primary alternative is to use positive end-

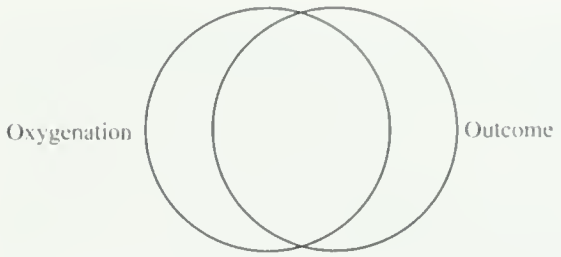

Fig. 5. Assumed relationship between arterial oxygenation and survival or other favorable outcome in managing acute respiratory distress syndrome (ARDS) patients: improving oxygenation signifies success in management and must surely improve outcome. This assumption was implicit in many studies of ventilator management of ARDS, in which increasing arterial partial pressure of oxygen was a management end point.

expiratory pressure (PEEP) and to attempt to lower F_{IO_2} by progressive increases in PEEP. We also have seen introduced pressure control ventilation, inverse inspiration-to-expiration ratio, decelerating inspiratory flow, and a variety of other manipulations of the ventilatory waveform in attempts to make the gas exchange better and improve oxygenation.

Then there have been the adjunctive measures for augmenting arterial oxygenation that have been introduced and subsequently studied in clinical trials, including prone positioning, inhaled nitric oxide, and extracorporeal life support or extracorporeal membrane oxygenation (ECMO). ECMO was studied in the mid-1970s in patients with severe ARDS and shown in a big multicenter NIH trial to offer no advantage in survival over standard management.[25] That study was even *repeated* by the NIH 15 years later, because ECMO advocates could not believe that the therapy was not effective, and once again a multicenter trial showed that ECMO produced no better survival than conventional ventilatory support, in this instance guided by a protocol.[26]

We now also have randomized, multicenter, controlled trials on prone positioning and inhaled nitric oxide. Inhaled nitric oxide[27] has been shown to raise P_{aO_2}, at least in most patients, but to have no effect on survival. And upcoming results of a large multicenter European trial of prone positioning in ARDS also found that most of the patients showed improved oxygenation but that there was no effect on outcome (L Gattinoni, unpublished data, 2001).

Contrast these findings to those of the ARDS Network study[14] showing substantially decreased mortality with low V_T conventional ventilation—in fact, using good old-fashioned assist-control. In the ARDS Network study, although measures of arterial oxygenation were ultimately the same in the 2 treatment groups, the ratio of P_{aO_2} to F_{IO_2} was actually lower, and patient exposure to higher F_{IO_2} greater, for the first several days in the low-V_T group. That is, the patients with better ultimate outcomes received higher F_{IO_2}

and had lower effective arterial oxygenation than the patients with larger V_T, who suffered higher mortality.

The above observations fly in the face of the conventional belief that the higher the F_{IO_2} the higher the risk of oxygen toxicity and a bad outcome. I think it's time we re-examined that belief. In my 25 years of attending in the medical and surgical ICUs at Harborview, I have become convinced that we have been sorely misled by an unwarranted fear of oxygen toxicity. In the Pacific Northwest, as in many cultures around the world, we have a myth of a giant forest beast called Bigfoot or Sasquatch. Well, I consider oxygen toxicity "the Sasquatch of the ICU." Everybody has heard of it and knows what it's supposed to look like, but the evidence of its existence is dubious at best!

I am well aware of the data from animal models and normal humans that demonstrate the toxic effects of breathing increased concentrations of oxygen.[28–30] However, I am not convinced that pulmonary oxygen toxicity really poses a clinically important risk in critically ill patients. Even with all the ARDS cases I've seen, I do not think I can identify a single case of oxygen toxicity. Maybe it happened and I didn't recognize it, which could well be the case, since pulmonary oxygen toxicity and ARDS are supposed to be a lot alike clinically. However, as I showed earlier, the mortality of ARDS decreased during a period when we were not making any substantial change in the oxygen concentrations we used. I also mentioned several studies that showed that short-term improvements in arterial oxygenation did not necessarily improve survival.

The ARDS Network study maintained F_{IO_2}, PEEP, and P_{aO_2} within specified ranges in order to minimize confounding variables, but there is no such uniformity in ordinary patient management. Managing ARDS may be as much about clinician behavior as it is about the patient's illness. If you don't believe that, just go from hospital to hospital in your own community and look at the differences in the way the same condition is managed. How high or low an F_{IO_2} clinicians are comfortable with and how much PEEP they will use in order to decrease F_{IO_2} below a certain level vary a great deal. Within the institution where I practice there are attending physicians who consider it acceptable to leave an ARDS patient on 70% oxygen and 10 or 15 cm H_2O of PEEP, whereas others will increase PEEP to 30 cm H_2O or more in order to get the F_{IO_2} down to 0.5 or less.

Similarly, the target P_{aO_2}, pulse oximetry saturation, or mixed venous oxygen saturation that triggers intervention in a patient being managed for ARDS varies considerably among institutions and among individual clinicians. In the absence of strict protocols or as a result of imprecise physician orders, it is often up to the nurse at the bedside to fine-tune the F_{IO_2} and maintain the various indices of oxygenation at individual "comfort levels." The "acceptable" level of arterial oxygenation varies from patient to patient and even for a given patient from shift to shift.

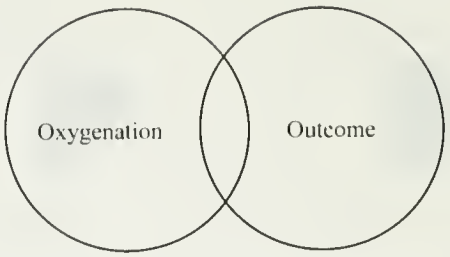

Fig. 6. Alternative relationship between oxygenation and outcome suggested by recent data from studies of nitric oxide and prone positioning in acute respiratory distress syndrome (ARDS): because relatively few ARDS deaths are due to inability to support oxygenation, improving oxygenation does not necessarily improve outcome.

Maybe the relationship between arterial oxygenation and outcome in ARDS looks more like Figure 6 than Figure 5. That is, maybe oxygenation and outcome are almost completely dissociated. In the studies of prone positioning (L Gattinoni, unpublished data, 2001) and inhaled nitric oxide,[27] there was no outcome benefit from interventions that increased P_{aO_2}. However, about 1 patient in 20 still dies because of insupportable oxygenation failure, so the concept illustrated in Figure 6 needs to show some degree of overlap, albeit probably much less than we have assumed in the past.

Does it really make a difference whether the P_{aO_2} in an ARDS patient is 50 or 100 mm Hg? That may seem a heretical question, and I can hear a chorus of responses from across the country saying, "Don't be ridiculous! Of course you shouldn't allow P_{aO_2} to be 50 mm Hg if you can make it higher than that." But I'm not sure we have much substantiation for the assumption that we should keep the P_{aO_2} in the normal range. Perhaps we should look more at oxygen content and delivery to the tissues and less at P_{aO_2} as the measurement to follow as we manage patients.[31–34]

Figure 7 shows the familiar oxyhemoglobin dissociation curve, but with oxygen content rather than saturation on the vertical axis.[31] A widely used version of this curve has oxygen saturation on the left vertical axis and content on the right, with a saturation of 100% corresponding to an oxygen content of 20 mL/dL of blood (same curve, different measures of the adequacy of oxygenation). But that is only the case when the hemoglobin concentration is normal at 15 gm/dL. If the patient's hemoglobin concentration is only 10 gm/dL, then the best oxygen content you can get, even with 100% saturation, is only two thirds as much. Similarly, for a hemoglobin concentration of 5 gm/dL, oxygen content would remain profoundly reduced despite a normal or even markedly elevated P_{aO_2}. Clinicians have known this for a long time, but sometimes

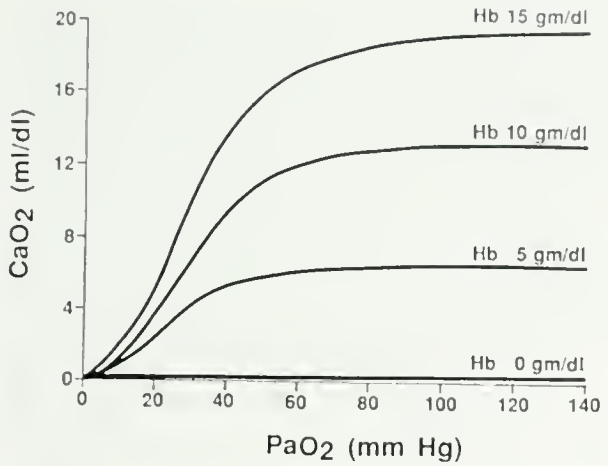

Fig. 7. The oxyhemoglobin curve for arterial blood, drawn with blood oxygen content (CaO_2) rather than saturation on the vertical axis, for hemoglobin concentrations of 5, 10, and 15 gm/dL. An arterial partial pressure of oxygen (P_{aO_2}) exceeding about 80 mm Hg results in a saturation approaching 100%, but the content of oxygen in the blood will remain reduced at hemoglobin levels below normal. Raising hemoglobin concentration from 10 to 15 gm/dL will increase CaO_2 substantially more than raising P_{aO_2} from 50 to 80 mm Hg. (From Reference 32, with permission.)

we don't apply it at the bedside as we should. We are going to need to pay much more attention to the other components of oxygen delivery to the tissues—hemoglobin level and cardiac output—in managing severely hypoxic ARDS patients.

I believe that in the coming years there will be less focus on increasing P_{aO_2} as a primary management goal. That is, we will use "permissive hypoxemia" just as we now use permissive hypercapnia in order to avoid complications of mechanical ventilation. You may remember that when permissive hypercapnia was first introduced into clinical practice, we were all pretty nervous about allowing P_{aCO_2} to get up to 50 mm Hg and arterial pH to get down to 7.30. Reluctant to subject our patients to acidemia when we had the means to avoid it, we did not let P_{aCO_2} rise above a certain level and we used bicarbonate infusions to keep the pH in a range where we were comfortable. Now, we manage ARDS patients with P_{aCO_2} in the 60s and 70s and even 80s without blinking, and we don't correct the pH when it gets down to 7.25 or even 7.20 if the patient is hemodynamically stable. The patients have not changed, but our approach to managing them has.

I think a similar transition needs to happen with respect to target P_{aO_2}. One of the things that's going to drive it is the fact that the measures we take to prevent and correct hypoxemia tend to cost us more than we may be able to afford. Here I don't mean just dollar expense, but also expense in terms of physiologic impact on the patient, particularly with the use of high levels of PEEP. I mentioned that I am not certain that I've ever seen oxygen toxicity as a clinical entity in the ICU, but I see the adverse effects of PEEP nearly every day—overt barotrauma from regional lung over-distention, and more invasive fluid support and monitoring to deal with the hemodynamic impact of raised intrathoracic pressure.

The future of mechanical ventilation for acute hypoxemic respiratory failure is lung-protective ventilation and the resulting permissive hypercapnia. I think the future will also be permissive hypoxemia, as we focus less on new ventilation modes and other manipulations to raise P_{aO_2} and more on adequate tissue oxygenation, on the prevention of complications, and on good general patient care. The "age of exploration" in mechanical ventilation is coming to an end, both because funding it has become prohibitive in both monetary cost and impact on the patient and because accumulating evidence suggests that we have been headed in the wrong direction.

Managing Acute-On-Chronic Ventilatory Failure

The management of acute-on-chronic ventilatory failure in acute exacerbations of chronic obstructive pulmonary disease (COPD) is another area that has evolved substantially and will continue to do so. The avoidance of endotracheal intubation will continue to emerge as a primary management goal, with more widespread and effective use of noninvasive positive pressure ventilation (NPPV) serving as the means for achieving this goal. There are already NPPV clinical practice guidelines and consensus statements,[35,36] and these are based on the highest level of clinical evidence: multiple randomized controlled trials with consistent findings.[37] However, my justification for including this subject in a discussion of the future of respiratory care is my impression that NPPV is still unknown, unused, and/or poorly practiced among many clinicians on the front lines of caring for COPD patients.

It is one thing to acknowledge that NPPV should be used in patients with acute-on-chronic ventilatory failure, but it is something else to carry it out effectively in the emergency department and ICU. Successful application of NPPV in the acute care setting requires both special knowledge and hands-on expertise, which do not come easily. I have seen this at my own institution, despite the presence of an excellent staff and several years of extensive education and training efforts.

In the future there will probably be more specialized areas of expertise, such as special respiratory units for managing patients with severe COPD. This has been the European model for many years, but in most areas of America it has been traditional for all critically ill patients to be managed in the same units. It may be that, at least with respect to obstructive lung disease, we will see more of these focused care units.

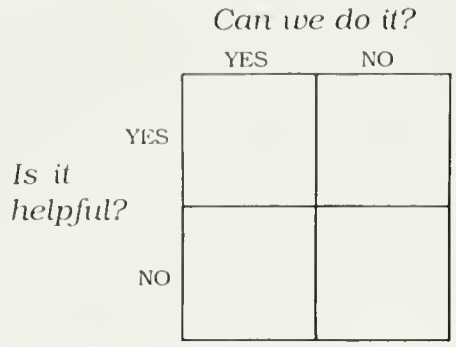

Fig. 8. Relationship between the technical capability of monitoring something and the clinical usefulness of the assessment. Monitors in the upper left-hand box are desirable, as would be those in the upper right-hand box that could be moved to the left. Procedures or devices in the lower left-hand box add risk, discomfort, effort, or expense without really providing additional scientific or clinical benefit. Unfortunately, if the future continues certain trends of the past, additional monitors not presently available may be added to the lower left-hand box from its counterpart on the right. (Reproduced with permission from Pierson DJ. Conference summary: pulmonary function testing: what, how, when, and why? Respir Care 1989;34(7):661–669.)

In managing COPD patients with NPPV, as in the care of patients with other conditions, both acute and chronic, we are going to see increasing use of protocols. Intubation, initial ventilator settings, ventilator adjustments, weaning, and extubation will all be guided by protocols, as I will discuss further below.

Monitoring the Critically Ill Patient

I would like to address monitoring in the ICU, as this is a subject that will—and should—change in the future. There is a clear need to do a better job of monitoring the right things while monitoring fewer of the wrong things. Examples of the right things to monitor include V_T corrected for circuit compression (something that is not even thought about at some institutions) and airway pressure measured at the distal end of the endotracheal tube rather than back in the ventilator circuit (where the effects of resistance in the system cause confusion). As I mentioned previously, we should monitor end-inspiratory plateau pressure rather than peak airway pressure to assess compliance and the risk of lung injury. We need to be capable of monitoring what the clinician really needs to know rather than what is more convenient to monitor (Fig. 8).[38]

The list of things that should be monitored should also include actual lung volumes, such as changes in functional residual capacity in response to changes in PEEP or other interventions, and the direct monitoring of intrinsic and total PEEP, rather than just the level set by the clinician. It would be very useful to have accurate, clinically relevant monitoring of the work of breathing, and particularly to be able to separate the work the ventilator is doing from what the patient is experiencing. I realize that there are systems out there that attempt to do part of this, but I would like to see a more direct measure of work rather than measures related to work in some way. Being able to directly monitor airway resistance in asthma patients would be very helpful.

We need not only to monitor the relevant changes in the patient but also to be able to do a better job of excluding artifact. As an example, consider airway pressures and oxyhemoglobin saturation. Peak airway pressure and end-inspiratory plateau pressure change when the patient coughs or rolls around in bed, and we need to be able to separate those changes from what happens as a result of a trend in the patient's condition, such as the development of pulmonary edema or bronchospasm. Artifactual and clinically irrelevant readings are major problems in oxyhemoglobin saturation, both with pulse oximetry and in mixed venous saturation monitoring via pulmonary artery catheter. In this and other areas, there is already considerable work being done to develop bedside monitors that will do a better job of filtering out extraneous and artifactual data and of giving us information we really need to see.

In the not-too-distant future, I believe we will succeed in making the connection between monitoring and management.[39] That is, there's a lot of talk about closed loop ventilation, wherein the measurements made by the monitors automatically adjust the ventilator, such as with automatic titration of F_{IO_2} in response to pulse oximetry readings or continuous P_{aO_2} measurements.

I believe there will also be—and should be—a trend toward monitoring more but displaying less. It would be very much like what has happened on the dashboards of cars. We no longer have gauges showing oil pressure, radiator temperature, or the amperage in the system. Instead, we know that those things are being monitored, and that if they get outside of accepted tolerances the system will let us know about it, but we don't have to be looking at the gauges all the time. I believe that something analogous to this is going to happen in ICU monitoring. There is a limit to how much information the human mind can keep track of. We need to have the capability to retrieve recent data when something does go wrong, but it will be the system that is monitored rather than its many components, at least from the standpoint of the clinician at the bedside.

Respiratory Care: The Profession

Definition and Characteristics

Now I would like to turn to respiratory care in the second context—respiratory care as a profession. The American Association for Respiratory Care (AARC) has defined this as "an allied health specialty, practiced with medical direction, in the treatment, management, control,

diagnostic evaluation, and care of patients with deficiencies and abnormalities of the cardiopulmonary system."[40] This profession exists primarily in the United States. There are now some dozen countries outside the United States and Canada with fledgling respiratory care professions modeled on the United States system, but predominantly this is still an American phenomenon.

An allied health profession separate from nursing, physical therapy, and clinical engineering was created in the United States a half century ago by a group of physicians and others who recognized the need for specialized expertise on respiratory disorders and the technology that was emerging to deal with them.

According to a recent survey commissioned by the AARC, the respiratory care workforce here is about 110,000 strong.[41] Seventy thousand of these professionals say that they are clinicians called respiratory therapists (RTs). We no longer have "respiratory techs" in this country: all people in the field are called RTs, and their credentials are either RRT (registered RT) or CRT (certified RT). The CRTT (certified respiratory therapy technician) credential has been superseded, as have 1-year training programs for entry into the profession. Now the educational standard is a 2-year training program, the product of which is the equivalent of an associate degree, and there are increasing numbers of baccalaureate and even graduate degree programs. In the last decade, along with this evolution of education and job designation, has come licensure, or at least official legal credentialing, of RTs in order to practice in nearly all 50 of the United States.

History

The history of the respiratory care profession is analogous to the history of pulmonary medicine and critical care: before about 1970 low-tech empiricism and individual ingenuity drove things. The ventilators were relatively simple, as I have mentioned, and there was little monitoring. ABG measurement was not yet in widespread clinical use. There were oxygen tents, and nearly everyone admitted to the hospital got intermittent positive-pressure breathing treatments. The respiratory therapy department was in the basement.

In the 1970s and into the early- and mid-1980s there was an explosion in respiratory care, just as there was in much of the rest of medicine. What had originally been known as inhalation therapy became respiratory therapy and finally, in 1986, was officially named respiratory care. It was a period of enormous technologic and economic growth. ICUs appeared, proliferated, and expanded, along with new ventilators, new modes, and new monitors. Hospital respiratory therapy departments became enormously profitable, and were no longer in the basement, literally or figuratively. Instead, they held a prominent position in the hospital because they were major revenue generators. Home oxygen therapy also burgeoned as both a clinical modality and a tremendous growth industry, as some half million Americans had oxygen systems installed in their homes.

Then came the 1990s, with continuing technical refinements but at a time of increasing cost constraint and reorganization. Ventilators became more technically sophisticated (microprocessor-controlled, with various combinations of the basic modes), but few all-new approaches were introduced. Hospital reimbursement shrank in the face of prospective payment, and respiratory care departments came to be thought of not so much as revenue generators but as cost centers, now an increasing burden on the institution. As a result, downsizing and reorganization affected a majority of the respiratory care departments in the country, and some were eliminated altogether.

Well, of course, the patients weren't eliminated when this happened. They and their needs were still there, and when the respiratory care department's central organization, skills, and expertise were dispersed throughout the hospital, it is not surprising that patient care and satisfaction deteriorated. Many of the hospitals that underwent these organizational changes during the 1990s have now taken steps to restore the previous structure and reassemble their departments.

At the same time that staffing was being cut back, with overall decreases in the quality and quantity of respiratory care available to patients in American hospitals, there was an enormous push to decrease length of stay and discharge patients earlier in order to reduce costs under prospective payment. This meant that patients were sent home from acute hospitalization earlier and sicker than before, a phenomenon with important implications for other parts of the respiratory care profession, as I will discuss further in a moment.

Evidence of the Effectiveness of Respiratory Therapists and Respiratory Care Protocols

As I implied earlier, assessment and management by protocol is where both respiratory care the profession and respiratory care the subject area are going. It has been shown that the use of protocols can decrease misallocation of respiratory care services, resulting in less expensive management. Rather than taking decision-making out of the hands of the doctors, protocols allow better utilization of the skills and time of both the physicians and the therapists.

There have now been 3 randomized controlled trials of ventilator weaning by RTs according to protocol.[42–44] A detailed review of these studies is beyond the scope of this article, but they involved large numbers of patients and all showed a shortened duration of mechanical ventilation without apparent deleterious effects. All the studies suggest that money can be saved when weaning is conducted

by way of treatment protocol driven by the clinicians who are at the bedside rather than having the doctor make all the individual decisions.

The effectiveness of RTs as consultants has also been reported in randomized controlled trials.[45,46] These studies show that use of protocols significantly decreases the number of patients receiving therapies that are not indicated and decreases the number who need therapies but do not get them.

A recent comprehensive review by Stoller summarizes the published documentation of the effectiveness of RTs.[47] This review uses the United States Preventive Services task force scheme for grading evidence of the effectiveness of therapies[48] to assess protocol-driven intubation, weaning, allocation of ABG measurement, placement of arterial lines, and performance of mini-bronchoalveolar lavage without bronchoscopy. Although the strength of the evidence varies, there is now considerable documentation of the value of RTs in the ICU setting.

Similarly, outside the ICU, the effectiveness of RTs in generating appropriate overall care plans, using protocols, has been shown at the highest evidence rating, with randomized controlled trials. Evidence in support of bronchial hygiene and oxygen titration protocols is close behind.

In ambulatory care, the strength of the evidence is not quite so good, but there is increasing documentation of clinical studies and institutional experience. Within the constraints of their study designs, these reports support the statement that the use of RTs in these settings, typically using protocols, improves care and is less expensive.[47]

Respiratory Therapists and End-of-Life Care

End-of-life care is another area that I think will see increasing attention in the realm of respiratory care in the coming years. RTs have been intimately involved with this topic for a long time, although they may not have acknowledged or realized it. After all, when life support is to be withdrawn, it is usually the RT who has to come to the bedside to actually carry out the plan. In the ICU and acute care hospital, but also outside, in rehabilitation and in home care, I believe that RTs will become increasingly involved in palliative care. The recent Respiratory Care Journal conference on palliative respiratory care[49] brought together internationally recognized experts and reviewed the subject in detail.

With respect to withholding and withdrawing maximal supportive intervention, I believe the future will bring more pre-ICU decision-making and earlier, more effective communication with patients and families about the interventions that may be undertaken in the ICU, considered in the context of the probable outcomes of those interventions. There is likely to be earlier determination of whether maximal supportive intervention is going to be continued, and this will be standardized and guided by protocols.

End-of-life and palliative care is an important component of what we do in critical care. We know that the majority of Americans who die in the ICU have life support withdrawn, rather than dying in spite of everything that we do. I believe we will see an increasing use of protocols for withdrawal of life support and protocols for assessing and managing patient distress while that is carried out. Such protocols are already in place in the institution in which I work.

Respiratory Therapists and Smoking Cessation

Smoking cessation and COPD prevention are another very important area of both respiratory care the subject area and respiratory care the profession. The United States Public Health Service recently issued an outstanding clinical practice guideline that is authoritative, practical, and covers all aspects of smoking intervention and smoking cessation. It is available at http://www.surgeongeneral.gov/tobacco/tobaqrg.htm and has also been published in Respiratory Care.[50] The National Lung Health Education Project has also recently called for the widespread screening of smokers by primary care providers in their offices in order to detect COPD.[51] Training these providers in the technique and interpretation of spirometry and in other areas pertaining to the control of smoking will heavily involve RTs.

In the future there will be increased and more effective efforts to get people to stop smoking. I believe we are actually going to make headway here. The war against smoking will have claimed more American lives than all of our military wars put together, but I believe that we will finally see that tide turn and that RTs are going to play a major role as that occurs.[52]

Respiratory Therapists and Home Care

As our population ages, the prevalence of chronic health problems affecting the respiratory system will continue to increase. I mentioned earlier that increasing pressure to reduce costs in the hospital has led to earlier discharge of patients and greater severity of acute illness at the time of discharge. This places an increasing burden on those who care for patients outside the acute-care hospital. Management may take place in long-term acute-care facilities, rehabilitation centers, skilled nursing facilities, or group homes, but most often it shifts to the patient's own home.

As older patients with more complex and severe illness are managed at home, there will be increased involvement of RTs and other nonphysician caregivers in the home. If this does happen it will probably reduce hospitalization and save money. However, for such a future to occur there

will have to be appropriate revision of existing reimbursement policies, which so far have not acknowledged the need for and contributions of RTs. In predicting this increasing deployment and effectiveness of RTs as physician extenders in the home setting, I have my fingers crossed because of the economic and political hurdles to be overcome. However, it definitely *should* happen if the best care is to be afforded our patients.

Respiratory Care: The Journal

Finally I come to the third use of the term *respiratory care*, which is the name of the journal Respiratory Care. Early in its 47-year history it was called *Inhalation Therapy* and it was small in size, scope, and frequency, published only every other month. Now monthly and different in many other ways, Respiratory Care is the peer-reviewed science journal of the AARC. It covers all aspects of respiratory care the subject area and the profession and was accepted for prospective inclusion in *Index Medicus* and MEDLINE as of the year 2000. Also, since an older database was assimilated into MEDLINE, you can also now retrieve via MEDLINE a substantial number of earlier studies published in Respiratory Care. The Journal has the largest monthly circulation of any peer-reviewed respiratory journal, and goes not only to the members of the AARC but also to individual subscribers, hospitals, and libraries. Its traditional RT readership is steadily expanding to include increasing numbers of physicians, nurses, and others involved in respiratory care the subject area. I believe that this will continue to increase, as will the numbers of high-quality manuscripts submitted for possible publication.

Conclusions

I said at the beginning that I would undertake the potentially foolish task of making 10 specific predictions about the future of respiratory care. I have mentioned each during the preceding discussion, but here they are in a list:

1. There will be less focus on raising P_{aO_2} as a primary goal in managing acute hypoxemic respiratory failure.

2. More attention will be paid to the adequacy of tissue oxygenation, irrespective of P_{aO_2}. With this "permissive hypoxemia," clinicians will tolerate lower P_{aO_2} and oxyhemoglobin saturation levels than they have previously.

3. We will have smarter monitors, with direct input into ventilators and other devices, but these monitors may not be indicating to us in real time everything that they are doing.

4. There will be more noninvasive ventilatory assistance and other focused interventions in acute exacerbations of COPD. Although this is the accepted standard of care already, it is not yet practiced widely enough or skillfully enough.

5. There will be more ICU triage and earlier determination of the appropriateness of maximal supportive intervention in the ICU, along with greater standardization of end-of-life care in that environment.

6. We will see the expanded use of protocols for assessment and management in all clinical settings pertaining to respiratory care. Although I have listed this one sixth, it should perhaps be number one, as the future of respiratory care in all 3 of its meanings lies with protocols.

7. There will be increased awareness of, expertise in, and resources for palliative care in all clinical settings. Respiratory care will be at the forefront as these increases take place.

8. There will be accelerating progress in smoking cessation and in the prevention and early amelioration of COPD.

9. RTs will assume a greater role as coordinators and care givers in home care.

10. Respiratory Care journal will continue in its role as science journal for the subject area and the profession, as the events forecasted above and its availability in *Index Medicus* stimulate increasing numbers and quality of submitted manuscripts.

Attempting to predict the future is almost always doomed to at least partial failure, but the preceding exercise will have been worthwhile if it has stimulated your thought and placed past developments into a context from which future developments can be understood and evaluated—however accurately they may correspond to my predictions.

Acknowledgments

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Books, Films, Tapes, & Software

Principles of Physiology, 3rd edition. Robert M Berne MD DSc and Matthew N Levy MD. St Louis: Mosby, 2000. Soft-cover, illustrated, 680 pages, $54.

I was especially interested in reviewing this book, as I have used Berne and Levy's *Circulatory Physiology* as a reference book for my cardiovascular physiology course for several years.

Principles of Physiology is divided into 8 sections, covering cell physiology, the nervous system, muscle, the cardiovascular system, the respiratory system, the gastrointestinal system, the renal system, and the endocrine system. The order of these sections is particularly appropriate, as the first 3 (cell physiology, nervous system, and muscle) address subjects that impact all the following sections. Thus, the reader learns the basis of muscular contraction before visiting a section in which muscle contraction plays an important part. Each section contains the requisite number of chapters to thoroughly cover that topic. Each chapter begins with a set of objectives, so the reader learns what the authors intend him to learn as he progresses through each chapter and knows at the beginning what the authors consider the most important concepts. New words are boldfaced and important concepts are set off in smaller capital letters. There are many colored boxes that provide more patient-specific information about general concepts that the text has just described. At the end of each chapter there are one or more case studies, with the answers provided at the back of the book. There is also a bibliography should the reader desire more information on a given topic. This paragraph actually sounds like the preface to this edition, wherein the authors describe the book's organization, making it the part of the book with which the reader should begin in order to obtain maximum benefit from reading it.

An interesting (and I am sure useful for them) technique that Berne and Levy used in producing this book was obtaining input from a panel of reviewers. As one would expect, 3 of the reviewers are faculty who teach physiology. However, as the vast majority of the audience for this text is composed of students, it makes great sense that the other 3 reviewers were medical students.

One item that the publishers seem to have gotten right (to my eye, at least) is the often troublesome printing of radiographs. In this book the reproductions are clear and the findings are readily discernible. Though a book of 680 pages is at first glance a daunting tome, it is highly unlikely that anyone would read this book from cover to cover. One would instead choose the sections that applied to the particular area of physiology in which one was interested. For instance, this book could easily serve as a text for both cardiovascular and respiratory physiology courses, reducing both the cost (an increasing concern) and the mass of textbooks each student is required to purchase and use. Its global utility also would make it less likely that the student would quickly unload the book at the end of the course. However, using this as a textbook would require more work on the part of the instructor, as besides the obvious section on the cardiovascular system or the respiratory system, he or she would need to pick the appropriate chapters from the more generic sections on cell physiology, the nervous system, and muscle.

This review may be starting to sound like an unreserved hymn of praise for this book. As with most texts, it certainly has its warts. However, these are more irritating than anything else and do not detract from the broad usefulness of the authors' efforts. The first section on cellular physiology is quite slow going, with an abundance of complex equations. It's probably mostly more than a practicing respiratory therapist would need. One irritant that immediately comes to mind is the frequent (on nearly every page) need to go to another page to look at a figure or table mentioned in the text. The reason for this, though, is the abundance of tables, figures, and graphs. Still, it would be nice to see these graphics on the same page as the text.

Another recurring problem is agreement of subject and verb. Three examples will illustrate:

"The excitability of fast—and slow—response fibers differ substantially."

". . . the hydrostatic pressure in the capillaries of the lower extremities are higher and that of capillaries in the head are lower. . . "

and

". . . systemic increases in the $P_{a\,CO_2}$ activates the sympathoadrenal system. . . "

The references to figures are sometimes confusing: the text mentions Figure 17–1A and 17–1B, yet there is no A or B in Figure 17–1. In another chapter, the text mentions a black line on a certain graph, but the legend for the graph discusses (and the reader can see) a red line and a blue line.

There are occasional confusing statements. At one point, they refer to the *ST interval*, during which the entire ventricular myocardium is depolarized, but then mention that the ST segment normally lies on the isoelectric line. When discussing the phases of the cardiac cycle and the events that occur during each, the authors insert the following statement: "During rapid ventricular ejection, left ventricular pressure slightly exceeds aortic pressure, and flow accelerates (continues to increase), whereas during reduced ventricular ejection, the reverse holds true." Lest you think you have misread the preceding, the next sentence goes on to say, "This reversal of the ventricular/aortic pressure gradient in the presence of continued flow of blood from the ventricle to the aorta. . . . " Of course, a fluid flows *down* a pressure gradient.

Another minor deficiency is the authors' seeming lack of clinical knowledge, as when they mention the patient's "tracheal catheter [being] temporarily disconnected from the respirator. . . . "

There is one set of case studies, after Chapter 19, in which the questions are phrased incorrectly for the first 2 questions, which will cause some head scratching about what the correct answer is—until you realize the misstatement. The first question is phrased, "What would cause a reduction in the patient's mean arterial pressure during the paroxysmal tachycardia?" with the correct answer, "A reflex increase in norepinephrine release from the cardiac sympathetic nerves." One quickly concludes that the question should have read "What would result from. . . ?"

Fortunately, the types of problems mentioned above are few and far between and do not detract from the usefulness of the

book as a reference text. For its price, this book could very easily displace any number of respiratory and cardiovascular physiology textbooks now in use. It would also be an excellent addition to the practicing therapist's library to provide review of concepts as they came up clinically.

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Pulmonary Pathophysiology. Gerard J Criner MD and Gilbert E D'Alonzo DO. Madison, Connecticut: Fence Creek Publishing. 1999. Soft-cover, illustrated, 418 pages. $27.95.

Pulmonary Pathophysiology is a textbook designed as a supplemental board review for second- and third-year medical students. It is one of 6 in a series of review textbooks by Fence Creek Publishing, and introduces concepts of pathophysiology in the context of clinical case studies. The text is divided conceptually into 3 sections. The first section, comprising Chapters 1 through 3, outlines the growth, development, structure, and function of normal lungs. It establishes a foundation of basic pulmonary physiology upon which the ensuing sections are based.

Chapters 4 through 8 compose the second section, which concentrates on pulmonary pathophysiology, emphasizing such concepts as the dynamics of air flow obstruction, the hemodynamic principles of pulmonary hypertension, the causes and sequelae of lung inflammation, and the mechanisms underlying respiratory failure and limitations to exercise.

The third section, consisting of Chapters 9 through 19, combines the concepts of the preceding 2 sections to focus on specific lung diseases, ranging from asthma to pleural disease.

This textbook is directed toward medical students who are transitioning physically as well as mentally from the classroom to the clinical arena; thus, the focus is appropriately on mechanisms of disease rather than on management issues. Nevertheless, respiratory therapists, nurses, and physicians-in-training may find this textbook useful. However, those well-versed in the contemporary management of respiratory disease will find the chapters covering specific lung diseases less informative. This is because statements of fact tend to be generalizations rather than evidenced-based citations of the current literature. For example, the chapter discussing lung cancer generalizes that "extent of disease at time of diagnosis" is a major prognosticator of survival, a description applicable to most cancers. Though factually correct, extending the emphasis to include details of the tumor-node-metastasis (primary tumor size, nodal involvement, and presence of metastasis) staging system for estimating prognosis would lend strength to the statement. Furthermore, adding such information would broaden the audience engaged to include residents and internists.

The authors provide a comprehensive review of pulmonary diseases, with special attention to pathophysiology, which is presented in a manner both easy and interesting to read. In fact, from the perspective of a medical student, one strength of this textbook is its organization and clarity of style. As previously mentioned, 3 sections compose the review: normal pulmonary physiology, common pathophysiologic processes observed in pulmonary medicine, and specific diseases. Each chapter outlines its contents and details the learning objectives prior to any introduction. A case study is presented early on and developed throughout the chapter, as salient principles of pulmonary pathophysiology are presented and fortified in the context of the current clinical process. This maintains the reader's interest and brings clinical relevance to each topic discussed.

Chapters 3 and 8, covering normal lung function and exercise patterns in disease, deserve special mention because they are particularly well written. Chapter 3 is subdivided into 6 sections and clearly and concisely details aspects of pulmonary physiology such as diffusion capacity and ventilation-perfusion relationships. Figures and their accompanying explanations illustrate and reinforce key points from the main text quite effectively.

Chapter 8 covers cardiopulmonary exercise testing and is outstanding for several reasons. It introduces, in easily understandable terms, fairly sophisticated concepts such as maximal oxygen uptake, anaerobic threshold, and other variables measured during exercise testing. The authors again make effective use of figures, accompanying explanations, and margin notes. In fact, the reader can glean the essence of the chapter's contents from them. Furthermore, there is a logical progression toward each learning objective from the beginning to the end of the chapter. Indications for and basic terminology of cardiopulmonary exercise testing are first presented. The authors next discuss how the various measurements made during cardiopulmonary exercise testing are used to interpret these studies and which measures are affected in different forms of exercise limitation. Finally, distinct patterns observed in obstructive, restrictive, and pulmonary vascular diseases are described and developed by the 3 clinical case studies presented.

The text is a paperback edition that is lightweight and portable. Its wide margins encourage notetaking as well as highlight salient concepts covered in the body of the text. This is a very visual book and contains numerous figures that are helpful in illustrating the ideas presented, which is one of the reasons **Pulmonary Pathophysiology** is so easy to read. The index is detailed but user-friendly. The incidence of typographical errors is low, but the few that do exist can inadvertently introduce incorrect information or omit important information. One example is in the discussion of steroid treatment of *Pneumocystis carinii* pneumonia. The text incorrectly states that a patient with an arterial partial pressure of oxygen *greater than* (rather than *less than*) 70 mm Hg should receive steroids. In addition, the section on parapneumonic effusions recommends instilling streptokinase through a chest tube for loculated pleural effusions that are suboptimally draining. The problem is that a typographical error omitted the word streptokinase, and this is only deduced from the unit dosages given.

The suggested readings that appear at the end of each chapter are appropriate for medical students and refer mostly to standard textbooks or reviews. Although rare in frequency, some of the information presented is dated. For instance, specific hypercoagulable states such as lupus anticoagulant syndrome and protein C and S deficiency are listed as risk factors for the development of deep venous thrombosis in the chapter on pulmonary embolism. However, more recent discoveries, such as factor V Leiden mutation, hyperhomocysteinemia, and the prothrombin gene mutation, are not included in this list.

In conclusion, **Pulmonary Pathophysiology** is highly recommended as a supplemental board review book for second- and

third-year medical students. It is recommended for respiratory therapists, nurses, or physicians-in-training who may also want a basic review of pulmonary physiology and pathophysiology. It is effectively designed as a study guide from its outline format, margin notes, excellent illustrations, and end-of-the-chapter questions. Overall, the authors have accomplished their stated aims of presenting pulmonary medicine with a focus on pathophysiology in a manner that is both instructive and enjoyable to read.

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Pulmonary Physiology, 5th edition. Michael G Levitzky PhD. New York: McGraw-Hill, 1999. Soft-cover, illustrated, 306 pages, $29.95.

Dr Levitzky points out in his preface that "this book is intended to be an introductory text." Though it works at that level, it is also reasonably comprehensive in its approach to the study of pulmonary physiology. Each chapter in the book could arguably be a text in itself, but the content is not glossed over and each topic is addressed in sufficient depth to make this a valuable resource. Each reader has to choose exactly how much detail he or she is looking for on each specific subject.

The text is divided into 11 chapters, with study questions and a useful appendix summarizing the basic tenets of pulmonary physiology. As in most general texts, Chapter 1 is devoted to the function and structure of the respiratory system. Though not in great detail, the content is accurate and well planned out. The electron micrographs of the lung parenchyma provide a good supplement to the drawing illustrations. Chapter 2 thoroughly describes the mechanics of breathing, and the illustrations are very helpful in guiding the reader. Compliance, elastance, and airway resistance are covered well, but additional depth in these topics could make this a more flexible text. The flow-volume tracings are a nice touch in addressing this subject.

Chapter 3, concerning alveolar ventilation, covers specific lung volumes and adds a little bit of pathology to enhance the content outline. The Bohr equation and the concept of physiologic dead space is handled well for the intent of the book. Blood flow to the lung is outlined in Chapter 4, in which the author explains the relationship between structure, function, and the control of blood supply within the pulmonary system; the explanation is better than in most introductory texts. It highlights the basic concepts and ties in the diagnostic mechanisms and pathology implications. The book also covers pulmonary edema better than most basic books, but it really doesn't lay an adequate foundation for the discussion of mechanical ventilation and positive end-expiratory pressure. A beginning student may have difficulty following those concepts. I would suggest a special chapter devoted to mechanical ventilation prior to the introduction of the topic.

Chapter 5 deals with ventilation-perfusion relationships. The author has paid great attention to detail in this portion of the book. Regional differences in ventilation and perfusion, as well as the physiologic consequences of their mismatching, are well integrated. The diagrams fully support the text and, indeed, make the content very easily understood. The concept of physiologic shunt and its calculation is also well laid out.

The next topic, in Chapter 6, is the diffusion of gases. This section covers the basic concepts of the diffusion of oxygen and carbon dioxide, and discusses measurement of diffusing capacity. Included at the end of the chapter is a list of the conditions that cause a decrease in diffusing capacity, but there is no discussion of the exact mechanism that creates the situation. More depth in the pathologic process would be helpful in this subject.

Next is the topic of oxygen and carbon dioxide transport in the blood (Chapter 7). This material is easily understand and covered in the right amount of detail. The content flows naturally and has a sound basis from the material covered in previous chapters. The gas laws are easily understood and, again, the diagrams support and enhance the text.

Chapter 8 is devoted to the body's regulation of acid-base balance, the chemistry of which is very clear and concise. Though many of these topics could be expanded on, the basic information is covered in sufficient depth for a newcomer to the study of pulmonary physiology. Likewise, clinical interpretation of blood gases is also covered reasonably well. I especially liked the coverage of the classification of the causes of hypoxia, which is not always covered in other texts. Chapter 9 describes the various mechanisms that control breathing. Table 9-1 provides a useful summary of the major reflex mechanisms and serves as a good overall view of the chapter content.

Chapters 10 and 11 outline several important specialized physiologic processes in pulmonary physiology. Chapter 10 deals with the nonrespiratory functions of the lung, such as pulmonary defense mechanisms, temperature regulation, air filtration, mucociliary clearance, and metabolism. The last chapter outlines how the normal physiologic mechanisms are disrupted or enhanced by conditions of stress. Specific subtopics include exercise, high altitude, and diving below sea level. I found this chapter informative and thought it a good wrap-up for the book. It forces the reader to refer back to the previous chapters to better understand the concepts of human physiology under stress, though I would have liked to have seen more on that subject. I would also like to pose the thought that disease also is a form of stress and that pathology of any nature affects the normal body physiology.

In summary, Dr Levitzky has written a very useful introductory text in the study of human pulmonary physiology. The design makes the text easily readable and not as tedious as many scientific texts. The illustrations are well thought out and enhance the salient points. The topics are well organized and follow a similar format to other texts on the subject. The chapter objectives match the content precisely and give the reader many good study guidelines. I found the content accurate and error free. The book provides basic information in a very uncomplicated way. I would highly recommend the text, but caution readers that many of its topics need enhancement in order to fully grasp the complexities of pulmonary physiology.

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Principles of Exercise Testing and Interpretation, Including Pathophysiology and Clinical Applications, 3rd edition. Karlman Wasserman MD PhD, James E Hansen MD, Darryl Y Sue MD, Richard Casaburi PhD MD, Brian J Whipp PhD DSc. Philadelphia: Lippincott Williams & Wilkins, 1999. Hard-cover, illustrated, 556 pages, $65.

Exercise intolerance is a common problem for patients with cardiac and pulmonary disease and is often associated with

impaired quality of life and poor prognosis. Cardiopulmonary exercise testing has become an important tool in the assessment and management of these patients and is now widely used in medical practice to quantify exercise limitation as well as to assess disease severity and response to treatment. A full cardiopulmonary exercise test with gas exchange measurement supplies detailed information about exercise performance and the interactions of cardiac, pulmonary, and peripheral muscle function during exercise. The correct interpretation of this information requires a comprehensive knowledge of normal exercise physiology as well as an understanding of the pathophysiology of certain medical conditions and how these conditions are likely to affect exercise performance.

Principles of Exercise Testing and Interpretation is the 3rd edition of the textbook regarded by many as the most comprehensive text available on interpretation and clinical applications of exercise testing. The authors have a vast amount of experience in the application of exercise testing in health and disease, having published an extensive amount of exercise physiology research from their own laboratories.

Chapter 1 introduces the reader to clinical exercise testing, with a brief overview of the book's content. It is followed by chapters focusing on the normal physiology of exercise, the pathophysiology of disorders limiting exercise, and the measurements collected during a standard cardiopulmonary exercise test.

Chapter 2 describes the normal physiologic and biochemical changes that occur during exercise, with a detailed focus on skeletal muscle function, lactic acid production, and the concept of the anaerobic threshold. The chapter ends with a discussion of the normal interactions between the pulmonary and cardiac systems during exercise.

Chapter 3 describes the variables measured during an exercise test and explains how these measurements can be used to understand the physiologic interactions between the lungs, heart, and skeletal muscles. Each measure is dealt with individually, with a clear definition followed by examples of the changes seen during normal exercise and abnormalities seen in certain disease states.

Chapter 4 discusses the pathophysiology of common medical conditions that limit exercise performance and how these conditions affect the variables measured during a clinical exercise test.

Chapters 5 and 6 discuss the equipment needed to perform clinical exercise testing and the normal values often used to interpret the exercise test. This is a particularly useful section of the book, outlining the mechanisms of exhaled gas analysis and cardiopulmonary monitoring as well as instructions on how to set up and perform a complete exercise test. The chapter on normal values contains a list of the authors' own recommendations, and references allowing readers to choose their own sets of normal values, if they wish.

The final 3 chapters deal with the interpretation and clinical application of exercise testing. Chapter 7 suggests a flow diagram for interpreting a cardiopulmonary exercise test, which is a useful guideline for those unfamiliar with clinical exercise testing. Chapters 8 and 9 deal with the clinical applications of exercise testing, with Chapter 8 describing common clinical settings in which exercise testing can be a useful diagnostic tool. Chapter 9 lists an extensive number of case presentations, with the typical abnormalities seen during a sample exercise test. This is an invaluable reference for those who report exercise tests, with detailed diagrams and text describing the characteristic changes for all the common cardiac and respiratory conditions. The book then finishes with an appendix containing summaries of symbols, a glossary of definitions, and calculations and formulae.

As a comprehensive review of exercise physiology, pathophysiology, and clinical exercise testing, this book is a tremendous success. The text is clear, up-to-date, well referenced, and supported by numerous diagrams to clarify some of the more difficult points. The authors do assume some knowledge of biochemistry and physiology, so it may be necessary to do some background reading, particularly in the early chapters on exercise physiology and pathophysiology. The text is well laid out and easy to follow, and as you progress through the book, it is clear that the chapters complement one another, leading to a comprehensive understanding of the role of clinical exercise testing in health and disease. One of the strongest points about this book is the fact that many of the chapters are self-contained. This makes the book an ideal reference tool for all individuals interested in exercise testing, allowing selective reading as necessary to gather specific information. This is further facilitated by a detailed contents section and index.

The intended readership includes physicians with an interest in cardiac and pulmonary disorders, exercise physiologists, and technicians, although the book is an excellent resource for anyone with an interest in exercise physiology and exercise testing. In particular, the chapters on setting up the exercise equipment, performing the exercise test, and interpreting the results supply sufficient information to set up an exercise laboratory without prior knowledge of exercise testing. To those with some prior knowledge of exercise testing, this book is an excellent reference text to troubleshoot most of the problems that can arise during testing and interpretation.

There are a few criticisms to be made. There is little information about the reliability and repeatability of the measurements collected during exercise testing, which is important when considering it in clinical practice. There is also very little mention of the problems associated with the assessment of ventilatory limitation, another common problem in clinical practice. Finally, the chapter on normal values could include a table of the alternative equations used to calculate predicted maximum oxygen uptake.

In conclusion, this book is an enjoyable and valuable read for all those with an interest in exercise physiology and pathophysiology. It is an excellent, comprehensive reference book describing the clinical application and interpretation of exercise tests and should be the text of choice in laboratories that carry out cardiopulmonary exercise testing.

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Measurement and Evaluation in Human Performance, 2nd edition. James R Morrow Jr PhD, Allen W Jackson EdD, James G Disch PED, Dale P Mood PhD. Champaign IL: Human Kinetics. 2000. Hardcover, illustrated, 383 pages, $55.

Measurement of human performance is the cornerstone of modern medicine. It allows quality research to be completed, and this leads to a better practice of medicine and more competent clinicians. **Measure-**

ment and Evaluation in Human Performance focuses on the fundamental concepts needed to conduct such research and describes how measurement is applied in a variety of settings related to human function.

Section I describes basic tools in measurement and evaluation: the nature of measurement, using microcomputers and statistics to analyze the data, and basic statistics. Section II discusses the concepts of reliability, validity, and grading. A separate chapter is presented in Section II on criterion-referenced measurement. This is an important addition for educators evaluating students in the health professions. Section III covers applications of measurement and evaluation to the classroom for teachers, and to the physical fitness lab for exercise physiologists and those interested in the evaluation of motors skills. One chapter in this section is of particular interest to educators: "Measuring Cognitive Objectives With Written Tests." This chapter deviates somewhat from the central theme of the text, but it is very well done and useful to educators writing and analyzing test items.

The intended audience of this text, according to the authors, is the "undergraduate student." They do not state which undergraduate students would use this text or with what course it should be used. This may explain why the book is written at a basic level and may make it more difficult for the text to become associated with a specific course.

I found this text very well written, easy to understand, and enjoyable to read. The authors have included numerous pedagogical features that assist the reader in comprehending the concepts under study: the list of key terms, chapter objectives, end-of-chapter reviews, and summary tables are helpful and very well done. The authors have also put together a Web site to help the student with important concepts. The Web site presents practice problems and more discussion of key concepts.

Another interesting feature of the text that I found very useful is the use of a case study that presents a measurement and evaluation challenge at the beginning of each chapter. The challenge presents a realistic problem that the reader probably will face in the future. Each chapter ends by returning to the case challenge and presents methods of approaching the problem using the concepts described within the chapter. This feature is unique and makes the content of the chapters much more pragmatic and valuable to the reader.

I found no typographical errors. The use of a second color adds to the visual appeal of the book and highlights key concepts and terms. It also calls the reader's attention to important tables and other important information.

The authors have done an excellent job of putting together a very readable text that even a novice of statistics and measurement would be able to read and comprehend easily. The layout of the text is appealing and adds to the quality presentation of the information within.

In spite of its positive qualities, I don't think this text has a place in an entry level respiratory therapy educational program. Educators today in entry level programs barely have enough time to present the essential medical aspects of respiratory care, so I doubt there will be time for this text. Educators who teach measurement in cardiopulmonary science programs at the bachelor of science or master of science level might adopt it. Students in advanced programs and research-oriented practitioners would benefit from reading this text. Even if they do not take a specific course in measurement, they will need to know the content of this text for research projects. The chapters on descriptive statistics, correlation and prediction, and inferential statistics would prove most useful to the respiratory therapist interested in an easy-to-understand basic overview of statistics. Educators would find the chapters on norm-referenced measurement, criterion-referenced measurement, summative evaluation, and alternate assessment very helpful in developing a better understanding of testing.

In summary, **Measurement and Evaluation in Human Performance** is a well written text that presents a wealth of information about the basics of research and the application of such concepts to measuring human performance. It uses numerous pedagogical features to assist the reader in learning the concepts presented. Though this book is beyond the curriculum of basic respiratory therapy programs, it could have a place in advanced programs at the bachelor of science or graduate school level. It would prove very valuable to practitioners interested in conducting quality research and to educators needing to learn more about measurement. I highly recommend this text to anyone who is looking for an easy-to-read text on statistics and measurement of human performance.

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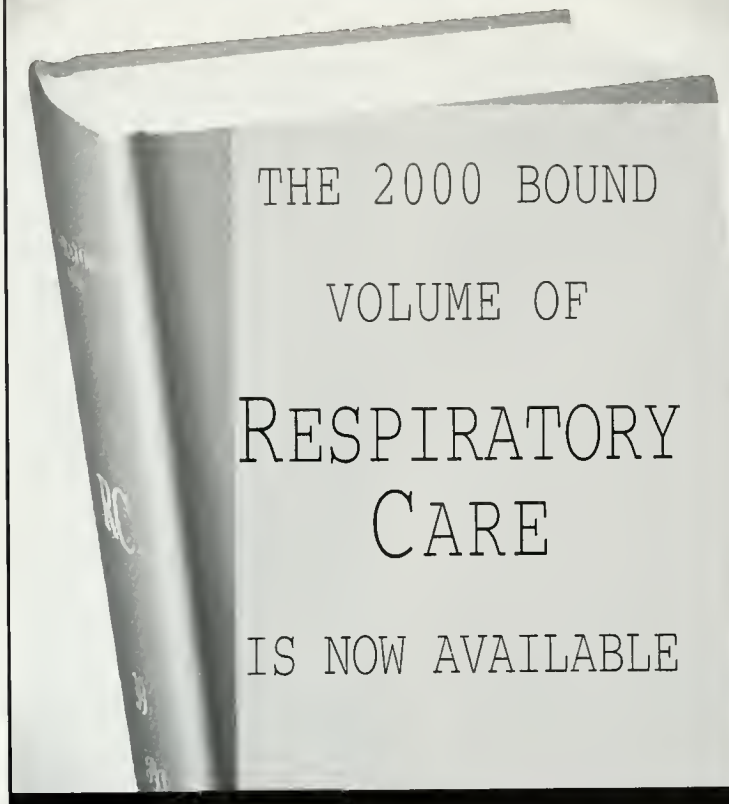

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American Association for Respiratory Care

MEMBERSHIP APPLICATION

Please read the eligibility requirements for each of the classifications in the right-hand column, then complete the applicable section. All information requested below must be provided, except where indicated as optional. See other side for more information and fee schedule. Please sign and date application on reverse side and type or print clearly. Processing of application takes approximately 15 days

- ☐ Active
- Associate
  - ☐ Foreign
  - ☐ Physician
  - ☐ Industriol
- ☐ Special
- ☐ Student

Last Name _____

First Name _____

Social Security No. _____

Home Address _____

City _____

Stote _____ Zip __ __ __ __ __ — __ __ __ __

Phone No. (_____) _____

Primary Job Responsibility (check one only)

- ☐ Technical Director
- ☐ Assistant Technical Director
- ☐ Pulmonary Function Specialist
- ☐ Instructor/Educator
- ☐ Supervisor
- ☐ Staff Theropist
- ☐ Staff Technician
- ☐ Rehabilitation/Home Care
- ☐ Medical Director
- ☐ Sales
- ☐ Student
- ☐ Other, specify _____

Type of Business

- ☐ Haspital
- ☐ Skilled Nursing Facility
- ☐ DME/HME
- ☐ Home Health Agency
- ☐ Educational Institution
- ☐ Manufacturer or supplier
- ☐ Other, specify _____

Dote of Birth (optional) _____ Sex (optional) _____

U.S. Citizen? _____ Yes _____ No

Have you ever been a member of the AARC? _____

If so, when? From _____ to _____

Preferred mailing address: ☐ Home ☐ Business

For office use only

FOR ACTIVE MEMBER

An individual is eligible if he/she lives in the U.S. or its territories or was an Active Member prior to moving outside its borders or territories, and meets ONE of the following criteria: (1) is legally credentialed as a respiratory care professional if employed in a state that mandates such, OR (2) is a graduate of an accredited educational program in respiratory care, OR (3) holds a credential issued by the NBRC. An individual who is an AARC Active Member in good standing on December 8, 1994, will continue as such provided his/her membership remains in good standing.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE

Place of Employment _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Medical Director/Medical Sponsor _____

FOR ASSOCIATE OR SPECIAL MEMBER

Individuals who hold a position related to respiratory care but do not meet the requirements of Active Member shall be Associate Members. They have all the rights and benefits of the Association except to hold office, vote, or serve as chair of a standing committee. The following subclasses of Associate Membership are available. Foreign, Physician, and Industrial (individuals whose primary occupation is directly or indirectly devoted to the manufacture, sale, or distribution of respiratory care equipment or supplies). Special Members are those not working in a respiratory care-related field.

PLEASE USE THE ADDRESS OF THE LOCATION WHERE YOU PERFORM YOUR JOB, NOT THE CORPORATE HEADQUARTERS IF IT IS LOCATED ELSEWHERE

Place of Employment _____

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City _____

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Phone No. (_____) _____

FOR STUDENT MEMBER

Individuals will be classified as Student Members if they meet all the requirements for Associate Membership and are enrolled in an educational program in respiratory care accredited by, or in the process of seeking accreditation from, an AARC-recognized agency.

SPECIAL NOTICE — Student Members do not receive Continuing Respiratory Care Education (CRCE) transcripts. Upon completion of your respiratory care education, continuing education credits may be pursued upon your reclassification to Active or Associate Member.

School/RC Program _____

Address _____

City _____

State _____ Zip __ __ __ __ __ - __ __ __ __

Phone No. (_____) _____

Length of program

- ☐ 1 year
- ☐ 2 years
- ☐ 4 years
- ☐ Other, specify _____

Expected Date of Graduation *(REQUIRED INFORMATION)*

Month _____ Year _____

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Demographic Questions

We request that you answer these questions in order to help us design services and programs to meet your needs.

Check the Highest Degree Earned

- ☐ High School
- ☐ RC Graduate Technician
- ☐ Associate Degree
- ☐ Bachelor's Degree
- ☐ Master's Degree
- ☐ Doctorate Degree

Number of Years in Respiratory Care

- ☐ 0-2 years
- ☐ 3-5 years
- ☐ 6-10 years
- ☐ 11-15 Years
- ☐ 16 years or more

Job Status

- ☐ Full Time
- ☐ Part Time

Credentials

- ☐ RRT
- ☐ CRT
- ☐ Physician
- ☐ CRNA
- ☐ RN
- ☐ LVN/LPN
- ☐ CPFT
- ☐ RPFT
- ☐ Perinatal/Pediatric

Salary

- ☐ Less than $10,000
- ☐ $10,001-$20,000
- ☐ $20,001-$30,000
- ☐ $30,001-$40,000
- ☐ $40,000 or more

PLEASE SIGN

I hereby apply for membership in the American Association for Respiratory Care and have enclosed my dues. If approved for membership in the AARC, I will abide by its bylaws and professional code of ethics. I authorize investigation of all statements contained herein and understand that misrepresentations or omissions of facts called for is cause for rejection or expulsion.

A yearly subscription to RESPIRATORY CARE journal and AARC Times magazine includes an allocation of $11.50 from my dues for each of these publications.

NOTE: Contributions or gifts to the AARC are not tax deductible as charitable contributions for income tax purposes. However, they may be tax deductible as ordinary and necessary business expenses subject to restrictions imposed as a result of association lobbying activities. The AARC estimates that the nondeductible portion of your dues — the portion which is allocable to lobbying — is 26%.

Signature _____

Date _____

Membership Fees

Payment must accompany your application to the AARC. Fees are for 12 months. (NOTE: Renewal fees are $75.00 Active, Associate-Industrial or Associate-Physician, or Special status; $90.00 for Associate-Foreign status; and $45.00 for Student status).

☐ Active	$ 87.50
☐ Associate (Industrial or Physician)	$ 87.50
☐ Associate (Foreign)	$102.50
☐ Special	$ 87.50
☐ Student	$ 45.00
TOTAL	$_____

Specialty Sections

Established to recognize the specialty areas of respiratory care, these sections publish a bi-monthly newsletter that focuses on issues of specific concern to that specialty. The sections also design the specialty programming at the national AARC meetings.

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☐ Education Section	$20.00
☐ Perinatal-Pediatric Section	$15.00
☐ Diagnostics Section	$15.00
☐ Continuing Care-Rehabilitation Section	$15.00
☐ Management Section	$20.00
☐ Transport Section	$15.00
☐ Home Care Section	$15.00
☐ Subacute Care Section	$15.00
TOTAL	$_____

GRAND TOTAL = Membership Fee plus optional sections $_____

- ☐ Total Amount Enclosed/Charged $_____
- ☐ Please charge my dues (see below)

To charge your dues, complete the following:

- ☐ MasterCard
- ☐ Visa

Card Number

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Signature _____

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Manuscript Preparation Guide

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Categories of Articles

Research Article: A report of an original investigation (a study). Must include: Title Page, Abstract, Key Words, Introduction, Methods, Results, Discussion, Conclusions, and References. May also include: Tables, Figures (if so, must include Figure Legends), Acknowledgments, and Appendixes.

Review: A comprehensive, critical review of the literature and state-of-the-art summary of a topic that has been the subject of at least 40 published research articles. Must include: Title Page, Outline, Abstract, Key Words, Introduction, Review of the Literature, Summary, and References. May also include: Tables, Figures (if so, must include Figure Legends), Acknowledgments, and Appendixes.

Overview: A critical review of a pertinent topic that has fewer than 40 published research articles. Same structure as Review Article.

Update: A report of subsequent developments in a topic that has been critically reviewed in RESPIRATORY CARE or elsewhere. Same structure as a Review Article.

Special Article: A pertinent paper not fitting one of the other categories. Consult with the Editor before writing or submitting such a paper.

Editorial: A paper addressing an issue in the practice or administration of respiratory care. It may present an opposing opinion, clarify a position, or bring a problem into focus.

Letter: A brief, signed communication responding to an item published in RESPIRATORY CARE or about other pertinent topics. Tables, Figures, and References may be included. The letter should be marked "For Publication."

Case Report: Report of an uncommon clinical case or a new or improved method of management or treatment. A case-managing physician must either be an author or furnish a letter approving the manuscript. Must include: Title Page, Abstract, Key Words, Introduction, Case Summary, Discussion, and References. May also include: Tables, Figures (if so, must include Figure Legends), and Acknowledgments.

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Test Your Radiologic Skill: A brief, instructive case report pertinent to respiratory care and involving imaging, including one or more radiographs or other images submitted as black and white glossy photographs that clearly illustrate the teaching points being made. Must include: Questions, Answers, and Discussion.

Preparing the Manuscript

Double-space the text and number the pages. Do *not* include author names, author institutional affiliations, or allusions to institutional affiliations anywhere except on the title page. On the Abstract page include the title but do not include author names. Begin each of the following on a new page: Title Page, Abstract, Text, Acknowledgments, References, each Table, each Figure, and each Appendix. Use standard English in the first person and active voice. Type all headings in initial-capital letters (eg, Introduction, Methods, Patients, Equipment, Statistical Analysis, Results, Discussion). Center the main section headings and place second-level headings on the left margin.

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Key Words. Include a list of 6 to 10 key words or key phrases in Research Articles, Reviews, Overviews, Special Articles, and Case Reports. Key words are best selected from the Medical Subject Headings (MeSH) used by MEDLINE and available at *http://www.nlm.nih.gov/mesh/MBrowser.html*.

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Hess D. New therapies for asthma. Respir Care (year, in press).

Article in a journal carrying pagination throughout the volume:

Legere BM, Kavuru MS. Pulmonary function in obesity. Respir Care 2000;45(8):967-968.

Article in a publication that numbers each issue beginning with Page 1:

Kallstrom TJ. Focus on asthma—disease management: a role for the respiratory therapist. AARC Times 1999;23(Oct):16, 17, 19.

Corporate author journal article:

American Association for Respiratory Care. Clinical Practice Guideline. Removal of the endotracheal tube. Respir Care 1999;44(1):85-90.

Article in journal supplement: (Journals differ in numbering and identifying supplements. Supply information sufficient to allow retrieval.)

Barnes PJ. Endogenous inhibitory mechanisms in asthma. Am J Respir Crit Care Med 2000; 161(3 Pt 2):S176-S181.

Abstract in journal: (Abstracts citations are to be avoided, and those more than 3 years old should not be cited.)

Volsko TA, De Fiore J, Chatburn RL. Acapella vs flutter: performance comparison (abstract). Respir Care 2000;45(8):991.

Editorial in a journal:

Giordano SP. What's that sound? (editorial) Respir Care 2000;45(10):1167-1168.

Editorial with no author given:

The perils of paediatric research (editorial). Lancet 1999;353(9154):685.

Letter in journal:

Piper SD. Testing conditions for nebulizers (letter). Respir Care 2000;45(8):971.

Book: (For any book, specific pages should be cited whenever reference is made to specific statements or other content.)

Cairo JM, Pilbeam SP. Mosby's respiratory care equipment, 6th ed. St Louis: Mosby; 1999:76-85.

Corporate author book:

American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group; 1977.

Chapter in book with editor(s):

Isono S. Upper airway muscle function during sleep. In: Loughlin GM, Carroll JL, Marcus CL, editors. Sleep and breathing in children: a developmental approach. (Lung Biology in Health and Disease, Vol 147, Claude Lenfant, Executive Editor.) New York/Basel: Marcel Dekker; 2000:261-291.

World Wide Web

American Lung Association. Trends in pneumonia, influenza, and acute respiratory conditions mortality and morbidity. February, 2000. http://www.lungusa.org/data. Accessed November 20, 2000.

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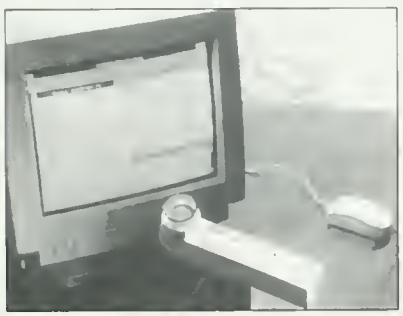
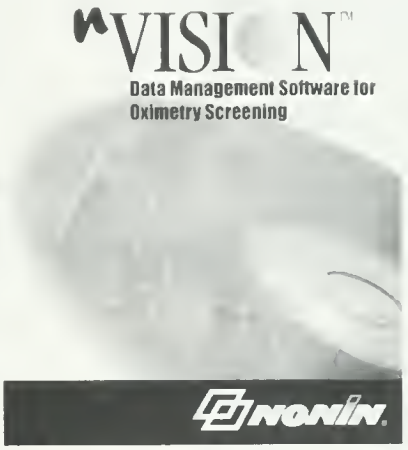


Spirometry System. FUTUREMED America Inc offers the SpiroVision-3, the newest in its line of PC-based PFT instruments. According to the company, the device is an accurate, inexpensive option for converting a PC or laptop into a complete spirometry system. FUTUREMED says the unit consists of a lightweight sensor that plugs directly into the serial port of a computer and that the Windows software interfaces with the sensor to perform FVC, VC, MVV, pre/post medication, and challenge testing. Company press materials say the unit's real time flow-volume loops and time-volume curves help therapists monitor patient performance for more accurate testing. Full-page printouts and colorful graphs let the user study individuals' tests or superimpose different test results for a comparison. For more information from FUTUREMED America Inc, circle number 165 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/



Overnight Oximetry System. Nonin Medical Inc has developed a Windows-based software package for overnight oximetry screening. The company describes nVISION as designed to provide easy viewing, professional analysis, simple report generation, and reliable data storage. Nonin says the oximetry data analysis tool increases productivity and decreases administrative costs and that the functionality of the new software makes it useful for most overnight screening situations where an accurate reading of blood oxygen saturation and pulse rate are needed.

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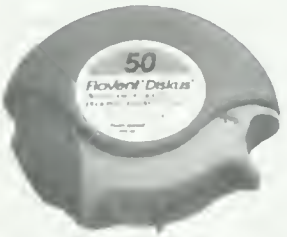

Inhaled Corticosteroid Powder Inhaler. The Food and Drug Administration has given GlaxoWellcome approval for the sale of Flovent Diskus (fluticasone propionate inhalation powder) for asthma patients 4 years and older. According to GlaxoWellcome, the new asthma treatment's inhaled corticosteroid may help reduce the airway inflammation that leads to asthma symptoms. Press materials describe the Diskus as a breath-activated powder delivery device that does not require hand-breath coordination, making it easier for children and other patients to use. The medication will be available in 50mcg, 100mcg, and 250mcg. For more information from GlaxoWellcome, circle number 167 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

Training Manikin. Armstrong Medical Industries Inc has introduced what it calls an easy and affordable Actar *D-fib* manikin for CPR and AED training. According to Armstrong, this new manikin carries the same qualities as the Actar 911 but includes added CPR features and new AED training capabilities. Company press materials say the CPR training features include jaw thrust, abdominal thrust, head tilt with closeable airway, realistic chest compressions, and all necessary landmarks. Armstrong also says the Actar *D-fib* is compatible with all training electrodes, and that electrodes don't interfere with chest compressions and can be easily removed. For more information from Armstrong Medical Industries Inc, circle number 168 on the reader service card in this issue, or send your request electronically via "Advertisers Online" at http://www.aarc.org/buyers_guide/

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July 21–23	Management and Education Sections, Summer Forum; **Naples, FL**	AARC, (972) 243-2272, www.aarc.org
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Aug. 14	Professor's Rounds 2001 Live Videoconference, Program 6	AARC, (972) 243-2272
Aug. 29–31	West Virginia Society for Respiratory Care's Respiratory Reunion; **Charleston, WV**	Jay Wildt at (304) 442-7474 or jay.wildt@mghwv.org; or Anna Parkman at (304) 357-4837 or AParkman@ucwv.edu
Sept. 11	Professor's Rounds 2001 Teleconference, Program 6	AARC, (972) 243-2272
Sept. 12–14	MD/DC Society for Respiratory Care, 20th Annual Conference by the Sea, **Ocean City, MD**	Joe Lynott at (202) 877-6086 or joseph.p.lynott@medstar.net; or Tom Striplin at (301) 784-5523 or tstriplin@mindspring.com
Sept. 12–14	Alabama Society for Respiratory Care's Annual Meeting, **Birmingham, AL**	Bill Pruitt, (334) 434-3405, wpruitt@jaguar1.usouthal.edu
Sept. 25	Professor's Rounds 2001 Live Videoconference, Program 7	AARC, (972) 243-2272
Sept. 26–27	MSRC Annual Meeting, **Sturbridge, MA**	Valeri-Ann Bolduc, (508) 429-7478, O2val@aol.com
Oct. 16	Professor's Rounds 2001 Teleconference, Program 7	AARC, (972) 243-2272
Dec. 1–4	47th International Respiratory Congress; **San Antonio, TX**	AARC, (972) 243-2272, www.aarc.org
Feb. 6–8, 2002	West Virginia Society for Respiratory Care's Winter Meeting, **Davis, WV**	Jay Wildt at (304) 442-7474 or jay.wildt@mghwv.org; or Anna Parkman at (304) 357-4837 or AParkman@ucwv.edu

Date	Other Meetings	Contact
Oct. 9–11	Cardiorespiratory Diagnostics 2001; **Las Vegas, NV**	Medical Graphics Corporation, Mari Orke, (800) 950-5597, ext. 444, www.medgraph.com
March 20–23, 2002	21st Annual Big Sky Pulmonary & Critical Care Medicine Conference; **Big Sky, MT**	American Lung Association of the Northern Rockies, (406) 442-6556, ala-nr@ala-nr.org

Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593

Scheduled Professor's Rounds 2001

Program #5 ARDS: The Disease and Its Management—Leonard D Hudson MD; Host David J Pierson MD FAARC—Video June 26 Audio July 17

Program #6 New Respiratory Drugs: What, When, and How—Joseph L Rau PhD RRT FAARC; Host Patrick J Dunne MEd RRT FAARC—Video August 14 Audio September 11

Program #7 Invasive Ventilation: The Latest Word—Richard H Kallet MS RRT; Host Richard D Branson BA RRT FAARC—Video September 25 Audio October 16

Program #8 Test Your Lungs-Know Your Numbers-Prevent Emphysema—Thomas L Petty MD FAARC; Host David J Pierson MD FAARC—Video October 23 Audio November 20

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http://www.aarc.org
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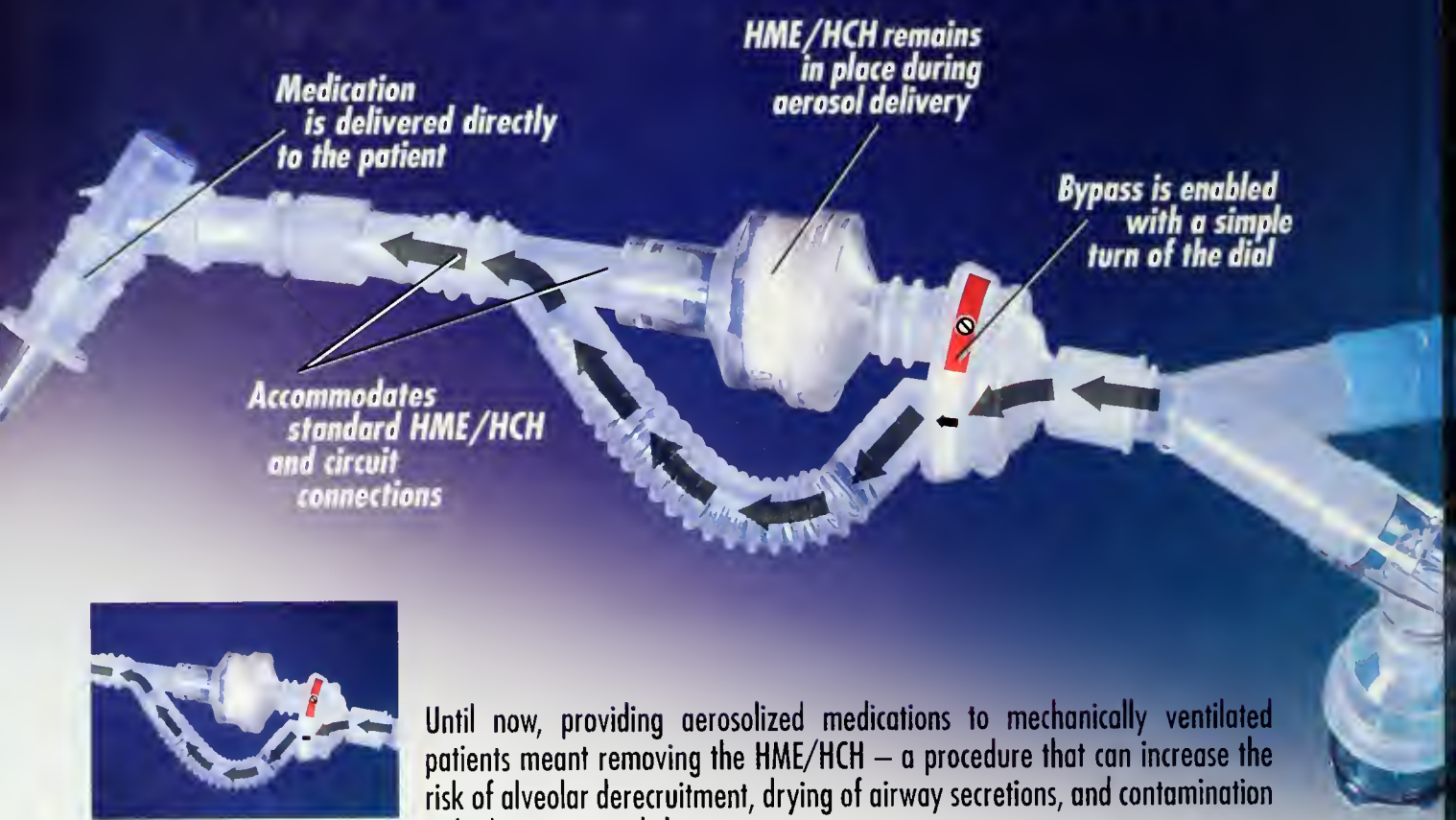
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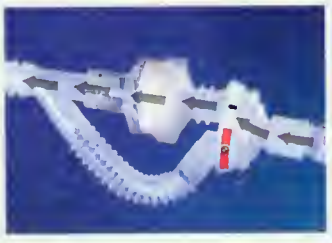

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